The Journal
OF THE FLORIDA MEDICAL ASSOCIATION

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Jerome A. Hilger

Program Medical District Meetings
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1. Michael, N.: Ohio State M. J. *48*:42, 1952.
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The Dermatologic Dangers of Sunlight

MORRIS WAISMAN, M.D.
TAMPA

The sun is the source of all life on the earth. Its pleasurable warmth and brightness are sought yearly by millions of people who come to Florida from many parts of the world. Throughout the year, by design or by the demands of occupation, tourists and resident Floridians bask in the solar radiance. Taken in moderation, sunshine offers many benefits which are too well known to enumerate. In excess, however, sunshine becomes a potent and dangerous agent, injurious to skin, eyes and internal structures. The range of safe dosage varies widely and is dependent on factors intrinsic in the skin and constitution of the person exposed as well as on the actual amount of actinic energy involved. It is the purpose of this paper to discuss some of the detrimental effects of sunlight both on the normal skin and the skin in pathologic states, as well as to point out means of attaining protection from biologically dangerous radiation.

A brief review of the position of the solar spectrum with reference to the electromagnetic system of radiant energy is pertinent (fig. 1). The effective range of radiation from the sun stretches from the ultraviolet to the infrared, from 2,900Å (angstrom units) to 30,000Å. The shorter wave lengths of sunlight are absorbed by the atmosphere, so that for practical purposes rays shorter than 2,900Å are not encountered on exposure. The region between 2,800Å and 3,100Å is characterized by intense biologic effects, antirachitic, antibacterial, and carcinogenic. Between 2,900Å and 3,100Å the erythemic action or burning effect upon the exposed skin asserts itself. From 3,000Å to 4,200Å, pigmentation is induced in the normal skin. Ordinary glass cuts off wave lengths less than 3,200Å, but transmits the longer wave lengths.

Among normal skins some are inherently able to tolerate only small amounts of exposure to sun without manifesting erythema and other evidence of injury. The red-headed complexion is most vulnerable, as is widely recognized, followed closely by the blond. The protection offered by the pigment of the brunet skin is exemplified in highest degree by the dark-skinned races. Not only do the lighter colored skins react to ultraviolet radiation more violently with acute burning, but also they show a greater proclivity to chronic damage and resulting aftermaths of serious importance. Nevertheless, the brunet skin is not immune to damage, acute or chronic, if the exposure is sufficiently potent, repeated and prolonged. Nor does pigmentation protect from injury in cases of pathologic hypersensitivity of the skin to the sun's rays.

Sunburn

The teenager dedicated to acquiring the swarthy melanosis which current vogue decrees to be beautiful, or the health faddist baking out his ills and hopefully intent upon absorbing quanta of dynamic energy from the sun, or the ordinary citizen serenely bared, toasting himself for sheer enjoyment of the warmth and indolence — all, in the blissful security of the conviction that "it's good for you," are often heedless of the warning that sunshine might be less than an unalloyed blessing. Often also, until the catastrophe of acute sunburn has been experienced, the burning capacity of sunlight is underestimated by persons exposed.

Because of the added absorption of rays reflected from sand and water, sunburn at a beach will be more severe than after the same length of exposure inland. Loosely woven clothing does not necessarily protect, as those who have been sunburned through a shirt will sadly testify. White fabrics protect less satisfactorily than brown,

Read before the Florida Medical Association, Seventy-Eighth Annual Meeting, Hollywood, April 28, 1952.

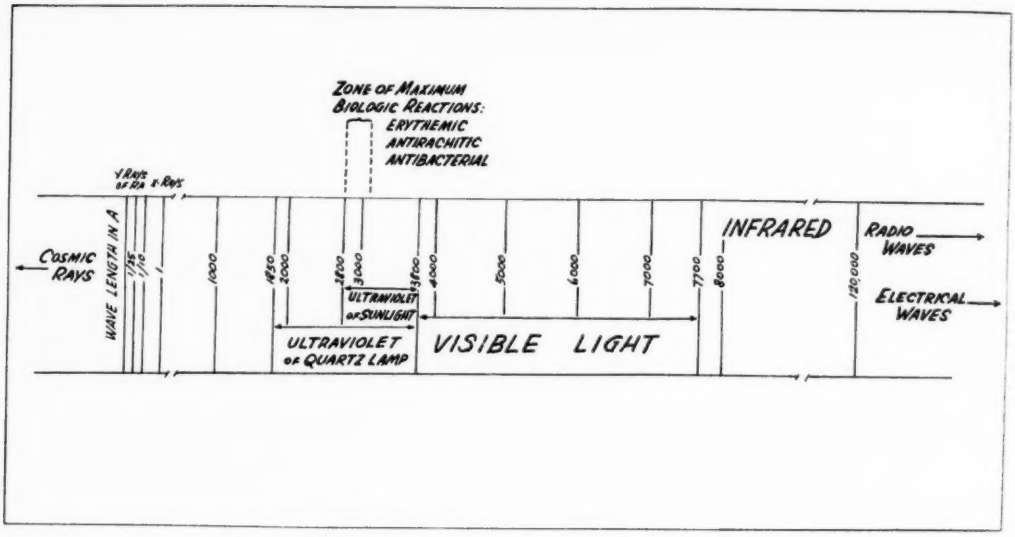

Fig. 1. — Schematic representation of the electromagnetic spectrum.

orange or red, and all fabrics protect less when wet.[1] Smoke absorbs the short wave lengths of sunlight and thus effectively screens, accounting for the paucity of ultraviolet light in industrial centers even in summer. Water vapor, however, allows these rays to pass, so that it is often possible to be badly sunburned on a cloudy day.[2] In Florida during a large part of the year, exposure to sunlight may be particularly hazardous between 11 a.m. and 3 p.m., when the solar rays fall almost perpendicularly. Small children tolerate only about half the adult erythema dose of ultraviolet.

The winter visitor to Florida who spends a few weeks or even a few months out-of-doors is in no great danger as a rule, because the total dose of radiation which he receives in a year is at most probably moderate in amount. This observation is particularly true if he takes pains to graduate his exposure to the sun so that optimal tolerance is achieved by his skin. The year-round resident, however, exposed daily to the maximum intensity of the sun, may suffer a considerable degree of cutaneous damage. Especially important is this possibility in the case of elderly people, whose skins are already vulnerable by virtue of senile changes present.

The natural protection of the skin against sunburn consists of a dual mechanism manifested by (1) thickening of the horny layer of the epidermis as a result of exposure to sunlight, and (2) melanin pigment developing in the epidermis.[2] The stratum corneum may thicken as much as sixfold in response to exposure to ultraviolet radiation, forming a mantle relatively opaque to the rays that cause sunburn. This is the principal method of achieving protection of the skin of white peoples, as distinguished from the melanin pigmentation which serves more importantly in the dark-skinned races. The sunburn spectrum (2,900Å to 3,100Å) results in formation of new pigment in the skin; but another source of pigmentation lies in the spectrum of longer wave lengths (3,000Å to 4,200Å), which results in the darkening of pigment already present in a "bleached" or colorless form. From these data it is evident that tanning can be induced without preliminary burning. The gonadal steroid hormones (estrogens and androgens) play a physiologic role in influencing pigmentation.[3]

The manifestations of acute sunburn need not be described. All are familiar with the fiery pain the wretchedness, the toxicity of the person who because of accident or ignorance or wilfulness has suffered an incandescent sunburn. Less familiar is the development of certain complications or sequelae of acute sunburn, which are enumerated: (1) Herpes simplex may appear, usually circumoral, representing the activation of dormant virus resident in the cells of the epidermis. (2) Folliculitis, occurring extensively over the sunburned parts, may develop as a result of hyper-

keratotic blocking of the sebaceous and sweat ducts. These structures are then predisposed to secondary infection because of the interference with free drainage of secretion from the ducts to the surface of the skin. (3) Vitiligo, the complete disappearance of pigment in areas of the skin, may be due to actinic destruction of the enzyme systems which regulate formation of melanin. (4) Chloasma, blotches of increased pigmentation, may first evolve after a sunburn and persist indefinitely, fading somewhat in the winter season but not disappearing completely. (5) "Showers" of pigmented nevi occasionally follow acute sunburn or accompany the subsequent tanning, particularly in children. (6) Persistent increased susceptibility to sunlight may result, representing a lowering of the erythema threshold, so that abnormally severe burning ensues upon re-exposure to relatively small doses of ultraviolet.

Prevention of Sunburn. Sunburn is easily preventable, and prevention may be attained by natural means or by the use of chemical and physical screens. The first exposure of the unadapted and unprotected skin must be short, and the time of exposure must be increased gradually as tanning and thickening of the epidermis proceed. In this way increasing tolerance can be safely developed.

By the judicious application of chemicals which absorb ultraviolet rays, sunburn can be prevented or minimized almost without regard to complexion or length of exposure. Chemical filters, effective in varying degrees, are: para-aminobenzoic acid and esters, phenyl salicylate, benzyl salicylate, menthyl salicylate, naphthol sulfonates, quinine and derivatives, esters of methoxy-coumaric acid, esters of anthranilic (ortho-aminobenzoic) acid, tannic acid and pyribenzamine. It is necessary to warn that any of these may be a cutaneous sensitizer, and para-aminobenzoic acid has even been detected in the anomalous role of a photosensitizer.[4]

Physical screens obviously bar the transmission of the sun's rays, and of these the wearing of adequate clothing is the most successful measure. Hats, gloves and umbrellas are of distinct help. Tying a handkerchief across the lower half of the face when in the sun, or sticking a slip of tissue paper across the lower lip, may avert serious solar injury to the lip. Effective also are face powders (titanium dioxide), zinc oxide, zinc stearate, bismuth subcarbonate, yellow petrolatum and red veterinary petrolatum. Proprietary baby oils which are commonly employed as suntan lotions have little if any antiburn value; and the practice of introducing iodine adds nothing to the sun-screening capacity of the oil.

A great number of sunburn preventives are marketed commercially. Some are most effective; many are inadequate; and a few of them indeed give no protection whatever.[5] To maintain effective shielding against sunburn, the preparation must be reapplied after an hour or two of exposure and after each swim. In hot weather, when perspiration dilutes and disperses the suntan agent, more frequent applications are of course necessary.

Satisfactory sunburn preventives may be prepared as follows:

Cream

Para-aminobenzoic acid 15 per cent in Ruggles' vanishing cream or other greaseless base. (Formula of Rothman and Henningsen.[6])

Lotion

Isobutyl-para-aminobenzoate	5 per cent
Alcohol	60 per cent
Glycerin	10 per cent
Water	25 per cent

The addition of 2 per cent tanic acid will help to simulate suntan and increase the sunburn-preventive action. (Formula of Schwartz and Peck.[7])

Farmer's Skin (Chronic Solar Dermatitis) and Skin Cancer

In persons exposed to sunlight over long periods of time — farmers, fishermen, cattlemen, building tradesmen, and devotees of outdoor sports and avocations — there develop prematurely senile changes of the skin (chataigne, or weatherbeaten, or farmer's or sailor's skin). The sandy or ruddy complexion which freckles readily is most predisposed to this change. The exposed parts become leathery, wrinkled, inelastic, freckled, telangiectatic, and spotted with precancerous keratoses (fig. 2a). The vermilion border of the lower lip becomes dry, fissured, pale and atrophic (solar cheilitis), and keratoses are likely to appear on its surface (fig. 2b). Degenerative changes of the connective tissue of the exposed skin lead to yellow discoloration, pebbling and deep wrinkling of the face and to the well-known rhomboidal furrows (cutis rhomboidalis nuchae) which cross-hatch the nape of the neck (fig. 2c). When chronic solar injury affects the backs of the

hands, multiple transient, small, irregular, purplish red petechiae may appear from time to time. Known as purpura solaris, the disease is probably caused by degeneration of the connective tissue and capillaries of the cutis.[8] So-called colloid degeneration of the skin is yet another, and rare, dermatosis in which solar injury of the connective tissue produces multiple yellowish translucent papules on the face and hands.

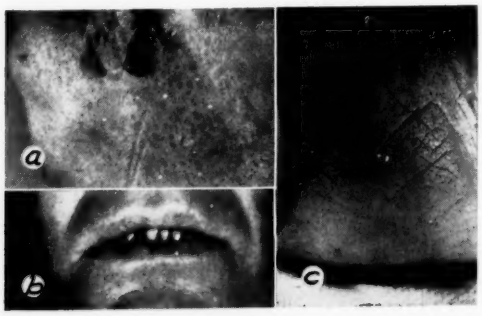

Fig. 2.—Chronic solar injury of the skin. (a) Chronic solar dermatitis (farmer's skin) showing multiple freckles and senile keratoses. Cancerous changes are already present in the crusted lesions. (b) Areas of pallor, keratosis and crusting in chronic solar cheilitis. (c) Cutis rhomboidalis nuchae: the typical quadrangular pattern of the sulci.

Cancers of the skin, the commonest of all malignant growths, predominate among outdoor workers of the white race, being practically absent in Negroes and Indians. Cancers of the skin, including the lip, occur much more commonly in the Southern part of the United States than in the Northern part.[9] It is safe to say that the incidence in Florida is at least five times that in Northern states. Moreover, the development of cancer of the skin among young adults (second, third, and fourth decades) in the South far exceeds the frequency of these neoplasms in the same age groups in the North.

Lesions appear chiefly on the exposed portions of the skin, the face, neck, and hands. Blonds are subject to skin cancer more than brunets, and redheads have the greatest susceptibility of all. The majority of patients with cancer of exposed surfaces of the skin have eyes of light color, that is, other than brown. Hall[10] has shown that the more brown-eyed inheritance a person possesses, the better protected he is from the carcinogenic rays of the sun. Blue-eyed descendants of blue-eyed parents are most susceptible as a group. Undoubtedly the brown-eyed skin averages a thicker stratum corneum, which accounts in large measure for its resistance to carcinoma. The wave lengths responsible for the formation of cancer are of the same spectral range as those of sunburn. Experimentally, it is possible to produce malignant tumors in the skin of white mice and rats by exposing them to sunlight or to radiation from a mercury arc lamp.

Carcinomas of the skin, both basal cell and squamous cell, develop upon chronically sunburned skin. It has been shown that 20 per cent to 25 per cent of senile keratoses in time eventuate in epitheliomas of the squamous cell type.[11] When it is considered that annually in the United States some 3,000 persons die of cutaneous malignant disease, the seriousness of the problem becomes apparent. If greater effort were made toward education of the public regarding the influences which lead to precancerous and cancerous changes in the skin, much might be achieved toward reducing the incidence of the commonest form of cancer.

Another common lesion of the skin related to the effect of sunlight is the "senile freckle" or lentigo (fig. 3a). This superficial, pigmented macule appears usually on the face and backs of the hands and forearms and tends slowly to increase in size. It is generally harmless. Occasionally a malignant transformation occurs which results in the lesion known as lentigo maligna. Lentigo maligna is a melanoma, circumscribed in situ for a time, but with the distinct capability for growth and widespread metastasis characteristic of all melanomas.

Somewhere in this area of classification of pigmentary lesions associated with chronic solar exposure must be listed parenthetically the persistent punctate, pigmented, and telangiectatic patches on the sides of the neck and the "V" of the chest designated *dermatose du triangle sternoclaviculaire* by Brocq[12] in 1916 (fig. 3b). This abnormality is observed predominantly in women. It is benign and of cosmetic importance only.

An aspect of the subject of carcinogenic effects of solar radiation which is usually side-stepped concerns the relationship of outdoor occupation and the development of cutaneous malignant disease. There is no doubt that in many instances the causal relationship of job to cancer is clear. In other cases the connection is equivocal, obscure,

or complicated by such factors as the workman's outdoor avocations and solar damage to the skin from nonoccupational exposure in earlier life. Preemployment examinations, for screening out persons with skins unsuitable for outdoor work, will probably in time become essential. To establish pertinent principles of guidance for the physician and the insurer is a project which awaits thoroughgoing study. Sooner or later this issue must be faced realistically by industrial commissions and an equitable solution worked out; and almost certainly the same problem will have to be acted upon also by the military services and the Veterans Administration.

Fig. 3.—Pigmented lesions attributable to the effects of sunlight. (a) Lentigines of the hands and forearms. (b) Punctate pigmentation and telangiectasis of the neck.

Xeroderma Pigmentosum

A rare familial disease, xeroderma pigmentosum (fig. 4) begins early in childhood when the skin of the exposed parts, because of hypersensitivity to sunlight, becomes thickened and develops freckles, keratoses, telangiectases, angiomas, and white atrophic spots. Photophobia and ectropion of the lower eyelids appear as the disease progresses. Later, atrophic and degenerative changes take place, leading to features of far advanced senile skin, actually a severely exaggerated farmer's skin. Neoplastic changes follow, with the formation of multiple carcinomas, basal or squamous celled, sarcomas, melanomas and other growths, benign or malignant. The child ultimately succumbs to a metastatic carcinoma.

Fig. 4.—Xeroderma pigmentosum. Freckles and keratoses are widespread. Compare with figure 2a.

Xeroderma pigmentosum is a mendelian recessive characteristic, and the parents are often consanguineous. Patients are sensitive to ultraviolet rays especially in the sunburn range. Sunburn radiation, however, only precipitates or accelerates the lesions of xeroderma pigmentosum, for apparently the condition may develop even in the absence of such radiation. Nevertheless, much can be accomplished in retarding the onset and rate of malignant degeneration if the child is completely shielded against exposure to the sun.

Solar Urticaria

Solar urticaria is a manifestation of photosensitivity in which brief exposure to sunshine produces a wheal within a few minutes, followed by an irregular flare spreading outward to the unexposed skin. The reaction, accompanied by intense itching, recedes or disappears within a few hours. Two types of urticarial light hypersensitivity are recognized: (1) that due to wave lengths below 3,700Å, a rare form, in which passive transfer of the sensitivity is demonstrable, indicating the presence of a circulating antibody;[13] and (2) that due to wave lengths above 3,700Å (visible light), in which passive transfer cannot as a rule be achieved.[14] Two different mechanisms are therefore probably involved. The action spectrum for the second group usually lies in the violet-blue portion of visible light (4,000Å to 5,000Å); hence wheals will develop even after exposure to sunlight through window glass, and

fluorescent lamps may elicit lesions whereas tungsten filament lamps will not. Because chemical filters are ineffective, recourse must be had to physical screens for protection. In the treatment of solar urticaria, antihistamine drugs are occasionally of benefit.

Solar Eczema (Polymorphic Light-Sensitive Eruption)

In solar eczema, pale itching papules or nodules appear on exposed areas, especially the backs of the hands, the face and the neck (fig. 5a), or an eczematous eruption develops. At times pinkish, thickened, unpigmented plaques occur on the cheeks and the neck. The condition predominates in females. Usually the eruption presents itself during the early spring, but sometimes there is no seasonal remission of the lesions in Florida, as contrasted with parts of the country where winters are long. By summer solar eczema may heal entirely even though exposure to sunlight continues, only to reappear the following spring. Most cases are evoked by abnormal sensitivity to rays of the sunburn spectrum. A few cases, however, respond to longer wave lengths as shown by the formation of lesions when sunlight is passed through window glass and by the failure of chemical filters such as para-aminobenzoic acid to protect. Lamb and his associates[15] stated that fluorescent lights might provoke and prolong a light-sensitive eruption.

Hydroa Aestivale

Hydroa aestivale is manifested by bullae of various sizes containing clear or discolored fluid, confined to the exposed parts and sometimes accompanied by edema (fig. 5b). When familial, hydroa appears in boys during early childhood, usually in spring or summer. In nonfamilial cases girls predominate 2 to 1.[16] The active wave length seems to be about 5,000Å, within the visible spectrum.

Two types are recognized: (1) hydroa with porphyria, in which blisters are large, not umbilicated, and attacks recur throughout life, associated with necrosis of skin and scarring, and even destruction of parts such as the nose, ears, and fingers; (2) hydroa without porphyria, which presents fewer but umbilicated lesions, less scarring or none at all, and spontaneous halting of the disease after puberty. Successful treatment of hydroa with steroid and gonadotropic hormones has been reported.[17]

Porphyria

Porphyrins constitute an integral part of the molecule of certain respiratory pigments, notably hemoglobin, myoglobin and cytochrome. Porphyrins originate in the body during the process of hemopoiesis, and not from the destruction of hemoglobin. The familial metabolic error which results in abnormal kinds and amounts of porphyrins excreted in urine and feces is known as porphyria. It perhaps represents a persistence of fetal porphyrin metabolism. Three clinical groups are recognized, congenital, acute, and chronic porphyria.

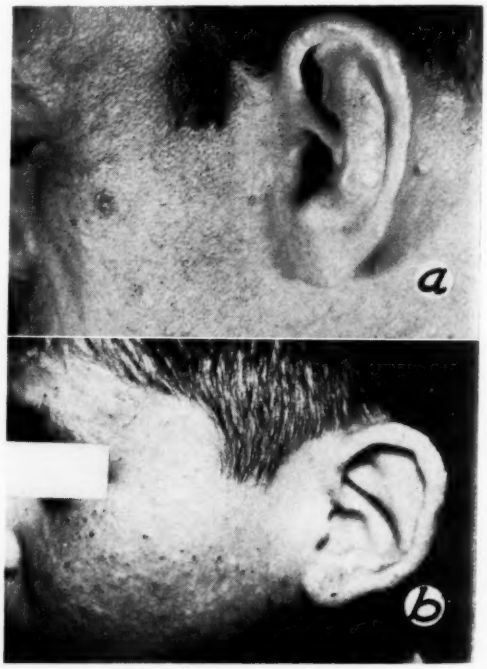

Fig. 5. — (a) Polymorphic light-sensitive eruption consisting of pale papules and discoid nodules. (b) Vesicles and crusts over face and ears of a boy with hydroa aestivale. (Photograph courtesy of Dr. Paul Fasal.)

Congenital porphyria is a chronic disease characterized by photosensitivity, with blistering and scarring of exposed parts. It appears at birth or early in life, predominantly in males, inherited as a mendelian recessive trait. Large quantities of porphyrin are excreted in the urine, to which may be imparted a dark red color. The teeth and bones may be discolored red by impregnated uroporphyrin.

Acute porphyria[18] is manifested by recurrent and alarming symptoms referable to the abdomen (nausea, vomiting, colic) or central nervous system (sensory disturbances, paralyses, convulsions, coma, psychotic behavior). The disease does not usually appear until the third or fourth decade of life. Occurring three times as frequently in females as in males, acute porphyria is transmitted as a mendelian dominant. Photosensitivity is rare.

Chronic porphyria presents increased porphyrin excretion, mild photosensitivity with a tendency to bullous lesions, and episodes of abdominal colic. Abnormal blistering after mechanical trauma may also occur. Sometimes pigmentation and hypertrichosis are evident. These cases possibly represent either congenital or acute porphyria in a mild form. Chronic porphyria may be present in a latent state, from which it can be stimulated to activity, usually in adult life, by damage to the liver from disease, alcohol, drugs, or other toxic influences.[19] In most cases the cause is indeterminate.

It will be readily appreciated from the foregoing that the relationship of porphyria to photosensitivity is far from constant. In fact, most patients with photodermatoses excrete normal amounts of urinary porphyrin. But more patients with photodermatoses have increased urinary porphyrin levels than those with other dermatoses. Porphyrins may ultimately be shown to be inactive associates or products of photosensitization reactions rather than their cause.[20]

Photosensitization by External Contacts and by Internal Drugs

A few chemicals have the ability to produce a dermatitis if their contact with the skin is followed by exposure to sunlight. The wave lengths which produce photosensitizing effects are those absorbed by the particular photosensitizer (Grotthus-Draper law). Photosensitization of this type is achieved by certain plant products and derivatives of coal tar and is characterized by the appearance of dermatitis with pigmentation.

In meadow grass dermatitis (originally described by Oppenheim in 1926) itching blisters in an irregular, streaked and criss-crossed pattern appear within 24 hours after a sun bath on a grassy field or after walking bare-legged in weeds (fig. 6a). They heal with pigmentation, which may persist for months (fig. 6b). Meadow grass dermatitis is probably not uncommon in the United States, and Oppenheim[21] suggested that some cases are mistakenly diagnosed dermatitis venenata due to poison ivy or other external irritant. Undoubtedly the precaution of sun-bathing on towels or blankets reduces the incidence of this eruption.

At least fourteen plants have been identified which are capable of producing photodermatitis of this kind, the majority of them belonging to the families Umbelliferae (parsnip, yarrow, angelica) and Rutaceae (common rue, gas plant, lime).[22] The photosensitizing chemicals responsible for the reaction are believed to belong to the group of furocumarins. These sensitize the skin for long ultraviolet waves, especially around 3,340Å to 3,660Å.

Berlock dermatitis, the most frequent form of photodermatitis, is caused by oil of bergamot present in cologne and perfume. Pigmented areas appear corresponding to the site of application of the liquid (fig. 6c). These lesions at times assume the configuration of a drop, due to running of the liquid after its application (hence the name berlock, or breloque, signifying a locket or ornament suspended on a chain, which is suggested by the characteristic linear appearance).

To Dr. Wiley Sams[23] of Miami belongs the distinction of recognizing, investigating and describing the commonest photodermatitis of Florida, which he showed was due to the oily fraction of the peel of the Persian lime (Citrus aurantifolia, var. Swingle). Contact with the peel, as in preparing limeade, followed by exposure to the sun, causes within 24 hours the appearance of erythema, often to the extent of severe sunburn with blisters, but associated with minimal discomfort or pruritus. A brownish discoloration, usually on the hands and forearms and occasionally on the face, gradually replaces the erythema (fig. 6d). Pigmentation may persist for as long as six months. Sams proved that the longer wave lengths of ultraviolet rays are involved, just below the zone of visible light (3,100Å to 3,700Å).

A similar photodermatitis has been reported among persons handling figs or coming in contact with the fig tree.

Coal tar pitch, heavy coal tar distillates, petroleum products and asphalt are photosensitizers, causing an erythematous dermatitis followed by pigmentation among men working with them out-of-doors.[24] Rays with longer wave lengths, rather

fluorescent lamps may elicit lesions whereas tungsten filament lamps will not. Because chemical filters are ineffective, recourse must be had to physical screens for protection. In the treatment of solar urticaria, antihistamine drugs are occasionally of benefit.

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Porphyria

Porphyrins constitute an integral part of the molecule of certain respiratory pigments, notably hemoglobin, myoglobin and cytochrome. Porphyrins originate in the body during the process of hemopoiesis, and not from the destruction of hemoglobin. The familial metabolic error which results in abnormal kinds and amounts of porphyrins excreted in urine and feces is known as porphyria. It perhaps represents a persistence of fetal porphyrin metabolism. Three clinical groups are recognized, congenital, acute, and chronic porphyria.

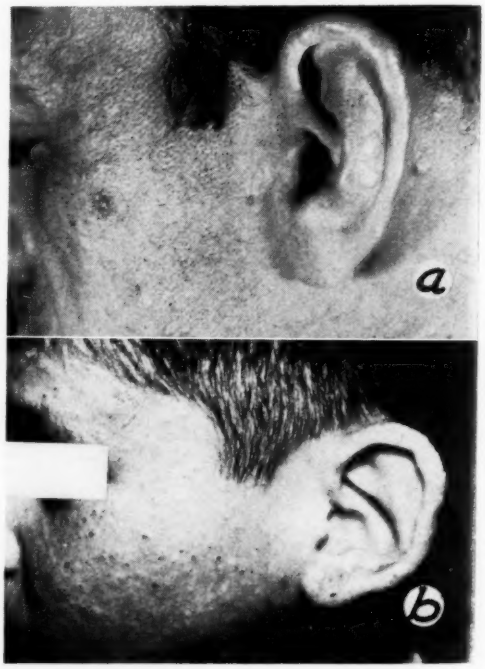

Fig. 5. — (a) Polymorphic light-sensitive eruption consisting of pale papules and discoid nodules. (b) Vesicles and crusts over face and ears of a boy with hydroa aestivale (Photograph courtesy of Dr. Paul Fasal.)

Congenital porphyria is a chronic disease characterized by photosensitivity, with blistering and scarring of exposed parts. It appears at birth or early in life, predominantly in males, inherited as a mendelian recessive trait. Large quantities of porphyrin are excreted in the urine, to which may be imparted a dark red color. The teeth and bones may be discolored red by impregnated uroporphyrin.

Acute porphyria[18] is manifested by recurrent and alarming symptoms referable to the abdomen (nausea, vomiting, colic) or central nervous system (sensory disturbances, paralyses, convulsions, coma, psychotic behavior). The disease does not usually appear until the third or fourth decade of life. Occurring three times as frequently in females as in males, acute porphyria is transmitted as a mendelian dominant. Photosensitivity is rare.

Chronic porphyria presents increased porphyrin excretion, mild photosensitivity with a tendency to bullous lesions, and episodes of abdominal colic. Abnormal blistering after mechanical trauma may also occur. Sometimes pigmentation and hypertrichosis are evident. These cases possibly represent either congenital or acute porphyria in a mild form. Chronic porphyria may be present in a latent state, from which it can be stimulated to activity, usually in adult life, by damage to the liver from disease, alcohol, drugs, or other toxic influences.[19] In most cases the cause is indeterminate.

It will be readily appreciated from the foregoing that the relationship of porphyria to photosensitivity is far from constant. In fact, most patients with photodermatoses excrete normal amounts of urinary porphyrin. But more patients with photodermatoses have increased urinary porphyrin levels than those with other dermatoses. Porphyrins may ultimately be shown to be inactive associates or products of photosensitization reactions rather than their cause.[20]

Photosensitization by External Contacts and by Internal Drugs

A few chemicals have the ability to produce a dermatitis if their contact with the skin is followed by exposure to sunlight. The wave lengths which produce photosensitizing effects are those absorbed by the particular photosensitizer (Grotthus-Draper law). Photosensitization of this type is achieved by certain plant products and derivatives of coal tar and is characterized by the appearance of dermatitis with pigmentation.

In meadow grass dermatitis (originally described by Oppenheim in 1926) itching blisters in an irregular, streaked and criss-crossed pattern appear within 24 hours after a sun bath on a grassy field or after walking bare-legged in weeds (fig. 6a). They heal with pigmentation, which may persist for months (fig. 6b). Meadow grass dermatitis is probably not uncommon in the United States, and Oppenheim[21] suggested that some cases are mistakenly diagnosed dermatitis venenata due to poison ivy or other external irritant. Undoubtedly the precaution of sun-bathing on towels or blankets reduces the incidence of this eruption.

At least fourteen plants have been identified which are capable of producing photodermatitis of this kind, the majority of them belonging to the families Umbelliferae (parsnip, yarrow, angelica) and Rutaceae (common rue, gas plant, lime).[22] The photosensitizing chemicals responsible for the reaction are believed to belong to the group of furocumarins. These sensitize the skin for long ultraviolet waves, especially around 3,340Å to 3,660Å.

Berlock dermatitis, the most frequent form of photodermatitis, is caused by oil of bergamot present in cologne and perfume. Pigmented areas appear corresponding to the site of application of the liquid (fig. 6c). These lesions at times assume the configuration of a drop, due to running of the liquid after its application (hence the name berlock, or breloque, signifying a locket or ornament suspended on a chain, which is suggested by the characteristic linear appearance).

To Dr. Wiley Sams[23] of Miami belongs the distinction of recognizing, investigating and describing the commonest photodermatitis of Florida, which he showed was due to the oily fraction of the peel of the Persian lime (Citrus aurantifolia, var. Swingle). Contact with the peel, as in preparing limeade, followed by exposure to the sun, causes within 24 hours the appearance of erythema, often to the extent of severe sunburn with blisters, but associated with minimal discomfort or pruritus. A brownish discoloration, usually on the hands and forearms and occasionally on the face, gradually replaces the erythema (fig. 6d). Pigmentation may persist for as long as six months. Sams proved that the longer wave lengths of ultraviolet rays are involved, just below the zone of visible light (3,100Å to 3,700Å).

A similar photodermatitis has been reported among persons handling figs or coming in contact with the fig tree.

Coal tar pitch, heavy coal tar distillates, petroleum products and asphalt are photosensitizers, causing an erythematous dermatitis followed by pigmentation among men working with them out-of-doors.[24] Rays with longer wave lengths, rather

than those of the sunburn spectrum, are responsible. Nonpigmenting photodermatitis of the lips may be produced by fluorescent coal tar dyes in lipsticks.

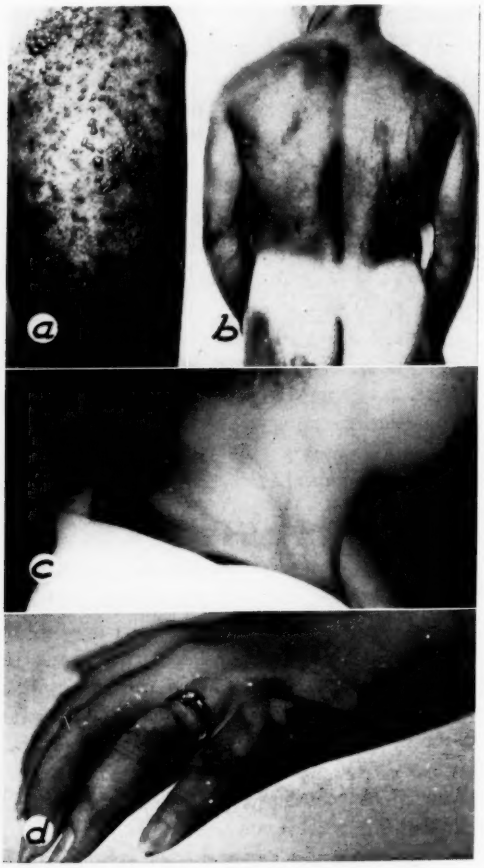

Fig. 6.— Photosensitization by external contacts. (a) Linear and criss-crossed bullae, and (b) striate pigmentation characteristic of meadow grass dermatitis. Observe the complete freedom of the area of the skin covered by bathing trunks. (Photographs courtesy of the late Dr. Maurice Oppenheim.) (c) Pigmented streaks on neck caused by application of cologne. The classical berlock or "running drop" effect is not present in this case. (d) Photodermatitis of fingers and hand from contact with oil of Persian lime.

Some fluorescent dyes (eosin, erythrosin, rose bengal, acridine dyes, hematoporphyrin) when injected into animals elicit, on exposure to sunlight, pruritus and edematous swelling of the face and ears, followed by necrosis of the skin in these areas. Occasionally barbiturates produce photoallergic effects. Sulfonamides are a relatively frequent cause of photosensitization, provoking an eruption which is usually morbilliform, becoming confluent and often associated with itching, fever and malaise. Less commonly present are edema, urticaria and vesiculation. Photodynamic contact dermatitis has been reported from the use of sulfanilamide ointment.

Photodynamic diseases occur in cattle, horses, sheep, goats and swine which have fed on certain plants before exposure to direct sunlight.[25] Similar photosensitization by foods has not been proved in man.

Diseases Associated with Sensitivity to Sunlight

Among certain diseases photosensitivity is presumed to be an inherent part of the morbid process. The relationship of the cutaneous lesions of pellagra to sunlight has long been recognized. In some cases, however, the distribution of the eruption is not in areas exposed to light; for example, the inguinal region may be affected. Apparently sunlight itself does not play the important role in pellagra that has been traditionally emphasized, and in many cases there is no unusual susceptibility to light.[2]

All types of lupus erythematosus often, but not always, become exacerbated on exposure to sunlight, and this exacerbation is particularly true of the acute and subacute disseminate types. The chronic discoid type, which is not associated with systemic manifestations or evidence of visceral disease, may or may not be aggravated by sunlight. Possibly the etiologic role of sunlight in lupus erythematosus has been excessively stressed. Many patients with chronic discoid lupus erythematosus suffer no evident harm from working in the sun, and they exhibit no improvement after exposure to the sun is eliminated. When exacerbation occurs in lupus erythematosus, the effective spectrum is probably in the sunburn band.

Acne vulgaris, acne rosacea and seborrheic dermatitis are occasionally made worse by exposure to sunlight (the adverse effect of concomitant summer heat is almost inseparably associated). So also are some cases of eczematoid eruptions (contact dermatitis, neurodermatitis, exfoliative dermatitis), "toxic" erythemas and erythema multiforme. Although not constant or predictable, the deleterious influence of sunlight is manifested sufficiently often to multiply the problems of dermatologic management of these diseases in tropical and subtropical climates. The effect of sunlight on the exanthem of smallpox is reputed

to be unfavorable. A papular photosensitivity eruption may accompany lymphogranuloma venereum. And finally, some cases of hyperthyroidism and pulmonary tuberculosis exhibit abnormal sensitivity to the sun, so that the patients may suffer considerable systemic reaction even with minimal burning of the skin.

Summary

This paper concerns itself with the detrimental effects of excessive sunlight on normal and pathologic skin. Variable degrees of susceptibility to ultraviolet radiation are exhibited normally, depending upon the complexion as well as upon the intensity and duration of exposure. Complications and sequelae of acute sunburn include herpes simplex, folliculitis, vitiligo, chloasma, pigmented nevi and persistent decreased tolerance to ultraviolet rays. Protection from sunburn may be achieved by graduated exposures to the sun, chemical filters and physical screens.

The development of so-called farmer's skin, or chronic solar dermatitis, is the most important effect of damage due to prolonged exposure of the skin to sunlight. Upon skin thereby altered, precancerous and cancerous lesions almost invariably appear. In emphasizing the carcinogenic role of ultraviolet rays, a plea is made for increased effort to disseminate an alarm against the peril of injudicious and indiscriminate exposure to the sun.

Uncommon and rare solar hypersensitivity dermatoses are xeroderma pigmentosum, solar urticaria, solar eczema and hydroa aestivale. These are described, together with some aspects of abnormal porphyrin metabolism. Much commoner are erythematous and pigmentary eruptions caused by plant products and coal tar derivatives if exposure to sunlight follows their contact with the skin. The chief entities in this category are meadow grass dermatitis, berlock dermatitis, lime oil dermatitis, and tar and pitch melanosis. Mention is made of the photosensitizing properties of some drugs, particularly the sulfonamides.

The relationship of photosensitivity to certain systemic diseases, notably pellagra and lupus erythematosus, is reviewed. Also noted is the deleterious effect of sunlight on dermatoses of the acneform, eczematoid and erythematous groups. In these cases the disturbing influence of the sun is inconstant, but it occurs with sufficient frequency to magnify the difficulties of dermatologic treatment in the subtropical climate of Florida.

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Discussion

Dr. Wiley M. Sams, Miami: The problems or disorders of the skin which we see following exposure to sunlight have been of interest to me for a number of years. I am happy, therefore, to have Dr. Waisman give us such an excellent factual account of these disturbances and review some of the more common ones which are rather frequently encountered in Florida, more so in this climate than in most places because of the large number of people who expose themselves to the sun. There is need for constant reappraisal of the dangers which may result from injudicious exposure to the sun because our state largely relies upon its climate and its sun for its attraction to tourists, and some of them incur difficulty during their sojourn, and sometimes they resolve not to return.

The person with a fair florid skin needs to be constant-

ly warned when he first exposes himself to the sun in Florida, and even then he fails to take adequate precautions in a good number of cases and receives a severe sunburn, or it may cause an exacerbation of some already existing dermatitis. We are perhaps less aware of many of the common factors in the ageing process of the skin which are due to repeated exposure and accumulation of injury from light over a period of years. Dr. Waisman has pointed out a number of these. I think the "turkey neck," the reddened neck which we see so frequently in fishermen and farmers, is recognized by all of us. Colloid degeneration of the skin, of which he had an excellent illustration, is not commonly recognized, but perhaps it is not so rare as we might suppose if we will keep on the alert for the changes in the skin that occur usually over the dorsum of the hands and the wrists.

The skin cancer problem in Florida and in the Southwest is more prevalent than in other parts of the country, and it reflects excessive exposure to sun. The general belief among most of our patients and most of our tourists that there is something which they absorb from the sun which rejuvenates them is, I think, the basis for most of the sun worship that we see. Actually, I believe that it is the rest and change of environment that lead to the improvement and not the effects of sun exposure.

DR. LEWIS CAPLAND, Miami Beach: In discussing a presentation, the discusser must find points of disagreement or points for amplification, or present some additions which might be of interest to the audience. I am unable to disagree with or amplify anything Dr. Waisman has said. So I thought I would add a few points which might be of interest to the audience.

The acute effect of sunburn on the peripheral circulation of the lower legs is an effect of immediate concern. By that I mean the consequences of sunburn on the lower legs of the older age group of the sun worshipers secondary to pronounced vascular dilatation. These effects are twofold: one is a dependent edema of considerable degree, and the other is a moderate to decided purpura and ecchymosis. Apparently the acute dilatation of the dermal blood vessels precipitates a latent varicose state which is unable efficiently to take care of the increased circulation. We see many patients with an edema which persists for ten or twelve days after the sunburn who can only be helped by ace bandages and bed rest. The other is the mild to great diapedesis occurring in the old blood vessels which are forced to dilate beyond their capacity; their fragile walls open up and let loose the red blood cells into the skin. The resultant purpura and ecchymosis are most disconcerting unless recognized and the patient reassured.

The next cases of interest are the results of the use of sunburn and suntan lotions, creams and oils. Here we have a group of cases which I have not been able to classify completely in my own mind. I have just pulled 200 records out of my files. I have crudely divided these cases into three groups: the urticarial photosensitivity group, the contact dermatitis group, and the contact type of photosensitization. The urticarial group occurs early after exposure to sun and application of oil. These cases are characterized by pruritus and urticaria. The patients must stay out of the sun, and when clear — and I say advisedly when clear, because a certain number of those in whom this urticarial reaction develops cannot return to sunbathing — may attempt sun exposure. This abnormal eruption may persist from two to six days. A certain number, as I said before, cannot reappear in the sun, and this limitation is most bothersome because we on the beach see people who come down only for two or three weeks at a time. They go immediately out in the sun, this eruption develops, and then their entire vacation is ruined.

The other contact dermatitis group comes from para-aminobenzoic acid, as Dr. Waisman has mentioned, and other materials in the suntan lotions and creams. These cases react and act like ordinary weed contact dermatoses, and of course the condition is the one with which we are all familiar. The third or contact photosensitization group gives the appearance of contact dermatitis, but is not related to a definite epidermal chemical sensitization.

DR. WAISMAN, concluding: I wish to thank the discussers. In closing, I should like to point out that the control of this problem of chronic solar dermatitis is essentially one of prevention, and since we are dealing with a condition which is undoubtedly caused by cumulative damage to the skin, I believe that it is our obligation to start the preventive program at the earliest possible age. In other words, our problem, I think, will be solved by starting the program of protection of the skin in childhood.

MEDICAL DISTRICT MEETINGS

Panama City, Oct. 27, 1952

Fort Pierce, Oct. 30, 1952

St. Petersburg, Oct. 29, 1952

Daytona Beach, Oct. 31, 1952

Exchange Transfusion in Erythroblastosis Fetalis

James J. Griffitts, M.D.
MIAMI

Erythroblastosis fetalis is a disease of infants caused by the specific action of maternal antibody on fetal antigen. The most important single determinant of damage to the infant in utero is the amount of maternal antibody which reacts with the specific antigen present in the infant's red blood cells.

At the Blood Bank of Dade County properly controlled maternal serum-antibody studies have furnished valuable diagnostic and prognostic information to the obstetrician and pediatrician. Data reported herein indicate the reliability of the information obtainable in the laboratory. The often expressed failure of the laboratory to provide prognostic information is due, in our opinion, to inaccurately performed laboratory procedures. There is a strong association between the level of maternal antibody and the degree of damage to the infant, a logical finding in view of the pathogenesis of this disease.

The understanding of the relation of maternal antibody titer to fetal and infant damage allows a rational approach to the management of the infant disease. While the details of the various blood antigens and antibodies are beyond the scope of this report, certain general features of the immunologic aspects of erythroblastosis fetalis will be presented. It must be stressed that the laboratory procedures involved in Rh antibody titrations must be properly controlled at all times in order to derive the valuable information which is available.

Case Data

The studies reported here represent 192 consecutive cases of immunized mothers bearing Rh positive infants. In the majority of cases serum specimens were collected at various intervals during the course of the pregnancy. In several instances the maternal antibody titer was determined at birth, the immunized status of the mother not having been previously recognized. Occasionally the first indication of the nature of the disease was recognized on the request for blood transfusion of the newborn infant. The procedures used in performing antibody titrations have been published elsewhere.[1] The practice of storing frozen specimens of serum withdrawn from the immunized mother during the course of her pregnancy has, more than any other single factor, led to consistency in antibody titrations. These specimens are restudied with later serum samples and serve as controls so that one may determine a definite change in antibody titer. Specimens of blood are studied within 24 hours after withdrawal, and the same red blood cells of known antigenic composition are used in all studies.

Infant Mortality Related to Maternal Antibody Titer

In spite of several excellent reports[2-4] on the relationship of concentration of the blocking variety of Rh antibody to the infant's disease, other reports indicate unreliability of maternal antibody level as a prognostic guide.

The relation of infant death to maternal antibody titer in 192 cases is shown in table 1. It will be noted that consistent mortality in infants appears only after the antibody titer has reached a level of 1:64, and that the mortality increases from this level upwards. Two deaths are recorded when the antibody titer was lower than 1:32, being 1:8 in both instances. These deaths are not explained by the data available since postmortem studies were not performed and laboratory data did not indicate severe anemia. There were 52 deaths in this series including 25 stillborn children. The mortality rate when the titer was less than 1:64 was 2.1 per cent. When the antibody

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titer was 1:64 or above, the mortality rate was 50.5 per cent. These figures indicate that the risk encountered by an Rh positive infant in an immunized mother is intimately related to maternal antibody titer.

Table 1.—Relation of Infant Death to Maternal Antibody Titer in 192 Cases; Last Prenatal Maternal Antibody Titer

	Trace-2	4	8	16	32	: :	64	128	256	512	1024	2048 and Higher
Number	8	8	24	23	30	: :	22	33	28	5	3	8
Deaths	0	0	2	0	0	: :	4	14	19	4	3	6
	2/93=2.1%					: :	50/99=50.5%					

Over-all mortality 52/192=27%
Excluding 25 stillborn deaths, per live child
27/167=16.1% mortality

Infant Morbidity Related to Maternal Antibody

Since anemia is a cardinal feature of the disease erythroblastosis fetalis, the necessity for blood transfusion is an accurate guide to the severity of the disease in a living child. The data presented in table 2 indicate that the need for transfusion was infrequent in cases in which the maternal antibody titer was 1:8 or less, but that it increased from this level upward. At a titer of 1:64 or higher, transfusion was indicated by the anemia and other manifestations of the disease present in every case.

Morbidity as represented by anemia requiring transfusion is intimately related to maternal antibody titer.

Table 2.—Relation of Maternal Antibody Titer to Need for Transfusion in 192 Cases

	Trace-2	4-8	16-32	64-128	256-512	1024 and Higher
Number	8	32	53	55	33	11
Living	8	32	53	46	21	7
Required transfusion	0	8	40	46*	21*	7*
Exchange performed	0	0	1	13	9	4

*Included are those dying before transfusion could be given.

Exchange Transfusion

Upon recognition of the relationship between maternal antibody and infant disease, indications for an exchange transfusion may then be established. The pathogenesis of erythroblastosis is the action of maternal antibody on fetal red blood cells. The maternal antibody crosses the placental capillary membranes and attaches onto specific antigens on the infant's red blood cells. The cells thus changed are more readily destroyed in the fetal tissues. The survival time of the red blood cells is probably inversely related to the amount of antibody fixed on them — the greater the amount of antibody the shorter the life of the cell. Anemia and the resultant anoxia lead to tissue damage. The compensatory efforts of the fetus to overcome the anoxia account for many of the features of this disease.

One is able by careful prenatal maternal antibody studies to establish the risk to which the infant is exposed. By prompt attention to the newborn infant, one may judge the need for transfusion and, in particular, the desirability of performing an exchange transfusion.

The removal of blood from an infant and its replacement with other blood was perhaps first performed by Hart[5] in 1925. Following the discovery and elucidation of the Rh blood factors, Wallerstein[6] specifically applied the procedure to erythroblastosis fetalis due to Rh incompatibility. Various technics have been developed by Wiener and Wexler,[7] Greenwalt,[8] Diamond,[9] Arnold and Alford[10] and others.

At the Blood Bank of Dade County the criteria for exchange transfusion in infants, based on laboratory procedures designed to measure the degree of damage caused by maternal antibody, are as follows:

1. The demonstration of human antibody fixed on the infant's red blood cells by the use of anti-human serum in the direct Coombs test. This test should be performed routinely on all specimens of blood sent to the laboratory with the request for transfusion of an infant in the first few days of life. Positive reaction to the test indicates that antibody is fixed on the cells of the child, shortening the life of the red blood cells in the infant's circulation. A quantitative estimation of the amount of antibody can be made with the slide test procedure which we employ at the Blood Bank of Dade County.[11]

2. The demonstration of anemia by an accurate red blood cell count and hemoglobin determination of the peripheral blood of the newborn infant. We have considered anemia to be moderate to severe with red blood cell counts below four million cells. This degree of anemia is usually associated with a strongly positive reaction to the Coombs test and with a maternal antibody titer in the range where mortality and morbidity are considerable. We have reserved all exchange transfusion procedures for such infants.

3. The demonstration of free antibody in the infant's serum. In our opinion the red blood cells of the infant will combine with maternal antibody to the degree that all red blood cell receptors for the antibody are completely satisfied. Frequently it will be found that considerable amounts of antibody remain free in the infant's serum. This is an indication that the red blood cells are heavily coated with antibody and are doomed to early destruction. In addition, the amount of free antibody present will be available for red blood cells which the infant may be able to produce so that the free antibody will further jeopardize the chances of survival of the infant.

4. Clinical manifestations of disease. The erythroblastotic infant may appear to be relatively normal at birth and within the course of the first few hours of life show rapidly progressing signs of anemia and jaundice. On the other hand, the infant may be pale and jaundiced at birth and show clinical signs of advanced disease as evidenced by bile staining of the umbilical cord, large liver and spleen, petechial hemorrhages, edema, and great lassitude. Clinical features are important in determining the severity of the disease. It is not infrequent, however, for the laboratory findings to disagree with the clinical impression shortly after birth. We have found that in instances in which the red blood cell count is low and the reaction to the Coombs test markedly positive, there is a rapid progression of the disease in spite of the early favorable appearance.

The laboratory procedures evaluating the infant's serologic status require at the most 15 to 30 minutes.

In accord with the criteria described, table 3 shows that exchange transfusions have been indicated and used only in infants of mothers with moderate or high concentrations of Rh antibody. Within any antibody titer grouping, individual variations of the severity of the disease are encountered, and in some of the cases, for example in the antibody level of 1:64, the infant may require only single or multiple transfusions; others may require exchange transfusions on the basis of the findings in the infant.

The purposes of the exchange transfusion are:

1. Supply the infant the large amount of blood required to restore a normal red blood cell count and hemoglobin.

2. Remove red blood cells coated with antibody, thus relieving the infant of end products of cell destruction which otherwise must be handled by tissues already overburdened.

3. Remove the excess and free antibody which otherwise will be attached to and damage new red blood cells produced by the infant.

4. Shorten the illness of the infant by giving cells compatible with the antibody passively acquired from the mother. The large single transfusion will reduce the number of transfusions subsequently required. If large amounts of antibody are present in the infant's plasma, transfusions may be required at intervals for as long as three months.[12]

The Technic of Exchange Transfusion

At the birth of an infant expected to have erythroblastosis fetalis it is recommended that a sterile plastic catheter* be inserted into the umbilical vein without delay. The catheter may be fixed in position with hemostats and clamped off while the necessary laboratory work is being performed. If a simple transfusion or an exchange transfusion is required, no time will have been lost, since the route of administration has been secured. If no transfusion is deemed necessary, the catheter may be removed and the cord tied.

The umbilical vein is large and readily identified in the cut end of the cord. Its tortuosity often necessitates the removal of the cord to less than 1 inch from the abdominal surface. Patience is required to insert the catheter to such depth (usually 3 to 5 inches) that a free flow of blood is obtained. Varying the position of the vein and catheter, and often stretching the umbilical vein, will be necessary to pass the catheter until a free flow of blood is established. Once the inferior vena cava is reached, the return of blood from the catheter will be prompt. The catheter may be clamped with a hemostat until the transfusion is set up. A 16 gauge intravenous needle inserted into the distal end of the No. 16 catheter provides an excellent channel for the administration and the removal of blood.

Occasionally the umbilical vein will not be available because of its extreme tortuosity or the possibility of early thrombosis. The recommended alternate route is a superficial branch of the saphenous vein exposed by a surgical incision in the femoral region.[10] Here it is recommended that the smaller sterile plastic catheter No. 19 be used. The end of the catheter may be cut so that a long bevel is formed; a small incision into the wall of the vein will then allow the catheter to be

*Polyethylene catheters Nos. 16 and 19, sterile and ready for use were obtained from Dade Reagents, Inc., Miami, Fla.

advanced from 3 to 5 inches into the inferior vena cava. The catheter should be tied securely in the vein with ligatures. This route has proved highly satisfactory for exchange transfusion.

The blood transfused should be as fresh as possible. It must be compatible from the standpoint of blood group and it must be of an Rh type which will not react with the specific Rh antibody present in the child's circulation. Most often the antibody is anti-Rh_0 and the blood transfused must be Rh_0 negative. The blood must be proved compatible by accurate crossmatch.

Universal donor blood (Group O) must not be employed for exchange transfusion in babies who are A, B, or AB. Group O blood contains anti-A and anti-B antibody usually in moderately high titer, and the use of large volumes of blood, as required in exchange transfusion, will load the infant's circulation with anti-A and anti-B antibody, which may have a deleterious effect on the infant's own A, B, or AB cells.

The transfusion is given as follows: A three-way stopcock is connected to a needle placed in the end of the catheter. The blood transfusion and administration set is connected to another adapter on the stopcock, and a syringe is placed in the third adapter. The syringe is filled with blood from the bottle, and 20 to 40 cc. of donor blood is slowly injected into the infant before blood is withdrawn. This gives the infant a small positive balance of blood in the beginning. Following the administration of blood, 10 to 20 cc. of blood may be withdrawn and collected in a test tube for further laboratory studies if desired. The syringe is again filled from the blood bottle and injected slowly into the infant. Alternate injections of 10 to 20 cc. of donor blood and withdrawal of like amounts of infant blood are continued. A careful record should be kept of the amount of blood injected and the amount withdrawn.

In most cases the administration of 500 cc. of whole blood and the withdrawal of 400 to 450 cc. is adequate to correct the severe anemia of the infant. The blood volume of an infant may be calculated as being 40 cc. per pound of body weight. In an infant, for example, weighing 6 pounds, the blood volume is approximately 240 cc. The injection of 500 cc. of whole blood with the removal of 460 cc. would be equivalent to an exchange of slightly over two times the infant blood volume. This amount is usually adequate, and we have been successful in the management of 2 severely affected erythroblastotic infants with such transfusions.

Blood is injected and withdrawn at a rate of not over 10 cc. per minute. Overload reactions are rarely encountered. It is recommended that the infant be maintained at a desirable body temperature and given oxygen as required. During the exchange transfusion 1 cc. of 10 per cent calcium gluconate may be given intravenously as required. This is designed to combat a temporary calcium shortage although the citrate in the donor blood is usually rapidly metabolized.

Results of Exchange Transfusion

Table 3 summarizes the experience with exchange transfusion, multiple transfusion and no transfusion among infants of mothers whose titers were 1:64 and higher. It will be noted that 70 per cent of infants given the exchange transfusion survived, compared to 80 per cent survival in infants given multiple transfusions. It must be realized, however, that when multiple transfusions were given, the severity of the disease in the infants was less than when exchange transfusion was indicated. In 5 mothers having extremely high titers of antibody the only surviving children were 2 who received exchange transfusions. In 7 cases in which no transfusion was given due to the late recognition of the disease, there were no survivals in infants of mothers whose titers were above 1:64.

Table 3.—Management of Erythroblastosis Fetalis According to Maternal Antibody Titer

	64	128	256	512	1024	2048 and Higher	Totals
Live births	20	26	20	1	2	5	74
Exchange transfusion	5	8	9	1	1	3	27
Survived	4	7	5	1	0	2	19
Transfusion	14	15	9	0	1	1	40
Survived	14	13	5	0	0	0	32
No transfusion	1	3	2	0	0	1	7
Survived	0	0	0	0	0	0	0

It will be noted also in table 3 that stillbirths occur with increasing frequency in mothers with high antibody titers. This observation has led us to the recommendation, on the basis of prenatal antibody studies and past history of incompatible transfusion or erythroblastotic children, that early delivery be performed in an effort to salvage some of the children. It is usually found necessary to perform exchange transfusion in these cases.

Summary

Erythroblastosis fetalis is due to the action of specific maternal antibody on the red blood cells of the infant.

The greater the amount of maternal antibody, the graver the risk to the infant in utero.

The exchange transfusion is a safe and rational procedure and should be carried out on the basis of certain criteria herewith presented.

Exchange transfusion, performed according to the severity of disease in the infant, improves the chances of survival.

Early delivery will conserve the lives of certain infants.

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Discussion

Dr. Lewis T. Corum, Tampa: I have enjoyed the fine presentation of Dr. Griffitts and I agree with his observations. I should like to stress the following points that Dr. Griffitts has presented, which I think are essential for the successful handling of cases of erythroblastosis:

1. The importance of following the Rh antibody titer throughout pregnancy. If this is done, one can anticipate with considerable certainty and security just what the problem will be at birth and can be prepared to carry out the necessary therapeutic procedures, namely, having on hand at the time of delivery a sufficient amount of Group O Rh negative blood to perform the transfusion. Sometimes it is rather difficult to find sufficient quantities of O Rh negative blood, particularly one chooses to use female blood.

2. Laboratory personnel present to perform the following tests, which are necessary for the diagnosis:

A. Direct Coombs test
B. Hemoglobin and red blood cell determination
C. Rh determination
D. Icteric index

3. Equipment and personnel available for carrying out the procedure, which, along with teamwork, is essential for a successful transfusion. For the six exchange transfusions we have performed in the past year, a team of five persons has been required, two doctors and three nurses, all of whom have been well oriented in the procedure. You cannot go over to the hospital, pick up an intern and two or three nurses who have never seen the procedure before and carry it out well.

In considering the advisability of early induction, one must weigh the dangers of prematurity against the possible damage that might be done to the infant by the continued action of the maternal antibody during the last three or four weeks.

In closing. I should like to ask Dr. Griffitts about the measurement of venous pressure during the transfusion, as has been advocated by others.

Dr. Lynn W. Whelchel, Miami: We have been most fortunate to have Dr. Griffitts give this paper on a subject which is of such importance to anyone having anything to do with obstetrics and pediatrics. In Dade County we usually take Dr. Griffitts as a matter of course and do not appreciate what we have, or rather do not stop to realize his help to us.

The pediatricians in this county, however, do recognize the value of his work and appreciate the help that he gives each individual when he has a problem of erythroblastosis. The reason I say that the profession as a whole fails to give him his just due is that we still have many newborns delivered in Miami for whom a call is put through to the pediatrician to come immediately to see the baby with whom there is something the matter. When the pediatrician arrives and evaluates the situation, he in turn puts in an urgent call for Dr. Griffitts. Studies are then initiated at this late date when they should have been in progress for many months previously.

While the pediatricians do appreciate the help offered, we do not believe that the problem is at all simple or that it is nearly solved. All of us have had cases of erythroblastosis in which, clinically, we thought the infant would do well, but from the laboratory findings we discover that there are great difficulties in store for the baby. Some of the babies on whom we have initiated an exsanguination transfusion die during the procedure or immediately afterwards. We cannot help but think that maybe we were wrong in undertaking the exsanguination transfusion, that the whole story has not been answered by the laboratory tests and, possibly, that the exsanguination transfusion is not without some danger in itself. Of course there are many cases in which the procedures to be carried out are definitely made clear by the laboratory findings and a great number of babies are saved, but we are always hoping to find methods to save many more.

In the discussion of the technic, I should like to stress again most strongly that one is dealing with newborn and frequently premature infants and that all precautions protecting the vital functions of these infants should be meticulously carried out during the time of study and during and following the transfusion. Nothing is gained by the transfusion if the baby succumbs from some other cause.

Dr. Griffitts: I want to thank the discussants, Dr. Corum and Dr. Whelchel. In answer to Dr. Corum's question about venous pressures, we have not made any observations although it would be extremely interesting and easy to do. We will try to add that to our studies. It might be a way in which one might relieve himself of the anxiety of thinking that maybe he had overloaded the infant, because a definitely increased venous pressure at any time in the procedure could be rapidly corrected, if it were an overload, by withdrawing blood.

Dr. Whelchel's concern about whether or not exsanguination transfusion should be carried out is warranted. Time did not allow to paint all the different shades of gray between white, when we do not have to transfuse, and black, when we know we have to transfuse. Biologic studies or studies of a living animal are fraught with all sorts of variables. We are certain that when the titer is high, we will encounter infant disease and have to treat it. To make a decision whether to perform an exsanguination transfusion, or simply to give the child a small transfusion and repeat it as necessary, is sometimes difficult. Personally, I tend to err a bit on the side of performing the exchange transfusion procedure. If one performs a single transfusion and then loses the baby, he will always have the question of whether he should have performed an exchange transfusion. The art of medicine is long.

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Acute Craniocerebral Injuries

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Patients with acute injuries of the skull and brain continue to be admitted frequently to every hospital where emergency cases are treated. The physicians on the staffs of such hospitals will be called on to treat these patients. Since a neurosurgeon will not be available, the general surgeon and family physician will be called on to treat them. There will be complications and associated injuries, which will require the services of specialists in every field of medicine. This subject should be of interest, therefore, to everyone present at this meeting.

Vital Signs

Whenever a patient is admitted to the emergency room of a hospital, one tries to determine his general condition and estimate the extent and degree of the injury. Frequently shock is not present unless the patient has associated injuries such as fractures of the extremities or spine, or severe thoracic and abdominal injury. Blood pressure, therefore, may be maintained at a normal level. With injury of vital centers, as the medulla or the brain stem, there may be a falling or lowered blood pressure. The state of consciousness may help to determine the severity of the brain injury. Also, the duration and degree of unconsciousness may have a bearing on the severity of the injury to this organ. With deep coma, when the patient cannot be aroused by any type of stimulation, there is probably a severe degree of brain injury. The size and reaction of the pupils are important, and a dilated, fixed pupil with deep coma probably represents a fatal injury. Pinpoint pupils with unconsciousness usually mean injury of the brain stem. A disturbance of the respiratory rate and rhythm with Cheyne-Stokes breathing probably means a severe or fatal brain injury. The same is true of a falling blood pressure with a rapid pulse rate. A subnormal temperature at first is expected, but within an hour or two the temperature rises, and a hyperthermia may develop with a severe and fatal injury.

Diagnostic Signs

Unconsciousness

Unconsciousness or amnesia of less than fifteen minutes' duration usually is associated with mild concussion of the brain. Of longer duration, for hours or days, it means a more severe brain injury. A patient is semiconscious when he can be aroused or move his extremities, while coma means no response to stimulation. A period of amnesia with apparent consciousness and a normal conversation may exist, and it may last for a considerable time. A retrograde amnesia may exist for a time prior to the accident.

Laceration and Contusion of the Scalp

A laceration of the scalp will locate the blow and aid in the examination for a fracture of the skull. Contusion of the scalp with hematoma beneath the pericranium may show a fluctuant swelling in the center with a raised ridge around the margin due to blood clots beneath the pericranium. This condition will give one a false impression of a depressed fracture of the skull.

Bleeding or Leaking Spinal Fluid from Ear and Nose

Bleeding from the ear, especially if there is spinal fluid escaping, means fracture of the middle fossa of the skull. Bleeding from the nose with escape of spinal fluid means fracture of the anterior fossa.

Pupillary Size and Reaction

A pupil of large size with reaction to light has little significance. A dilated, fixed pupil associated with other vital signs, however, may be of serious omen. Inequality of the pupils is of importance since a localized hemorrhage of the brain causing compression may produce a dilated, fixed pupil on the same side. This is so often seen with extradural and subdural hematoma.

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Cranial Nerves

Paralysis of the third cranial nerve will produce a dilated, fixed pupil, ptosis of the upper lid and extraocular paralysis except for downward and outward rotation of the eye. Paralysis of the sixth cranial nerve will cause an internal squint and signify an injury at the base of the skull. The seventh, or facial, nerve is frequently paralyzed by fractures in the middle ear and of the temporal bone. The facial paralysis will be of the peripheral type with all branches being involved. Injury to the optic nerve may be due to fracture through the optic foramen and may result in blindness. In such case, one should employ roentgenograms to rule out a depressed fracture of the optic foramens.

Paralysis

Paralysis or weakness of the extremities and of the lower part of the face may be associated with a severe brain injury. With an unconscious patient, one may obtain evidence of paralysis by observing the extent to which the face and extremities are moved by supraorbital pressure.

Reflexes

A hyperreflexia will be associated with a paralysis, but it must be borne in mind that with acute cerebral lesions due to trauma, especially while shock is still present, the reflexes may not be exaggerated. One expects to find the reflex involvement varying from a Babinski, Oppenheim or Gordon reflex to an ankle clonus on the side opposite the lesion and on the side of paralysis.

Convulsions

Convulsions do not occur frequently in the early stages of brain injury. Should a patient have a low convulsive threshold, however, it is not unusual for a convulsion to be associated with a slight blow on the head with mild concussion. On the other hand, should there be severe cortical brain injury with hemorrhage, either extradurally or subdurally, there may be a jacksonian convulsion. Likewise, a convulsion may be associated with a depressed fracture of the skull with an injured brain cortex, especially when it is near the motor area. When one sees convulsions in the early stages of brain injury, one should bear in mind that this condition may plague the patient for the rest of his life, and he will require adequate therapy to prevent the seizures after he has made an immediate recovery.

Blood Pressure, Pulse and Respiration

With increased intracranial pressure developing from an extradural hemorrhage or an acute subdural hematoma, there are apt to be changes in the blood pressure, pulse and respiration. For that reason they should be recorded every thirty minutes to an hour during the early stages of observation. Before the systolic pressure rises in acute intracranial pressure, there is likely to be a drop in the diastolic pressure. This gives an increase in the pulse pressure. Later on, the systolic pressure rises when the intracranial pressure exceeds that of the arterial pressure in the medulla. The pulse rate drops with increasing intracranial pressure as occurs with an extradural hemorrhage, and a pulse rate of 40 or 50 is not unusual. Should there be associated injuries, however, a slow pulse rate may not occur. The respiratory rate may not be seriously involved except in severe fatal injuries and in the late stages of increased intracranial pressure.

Hyperthermia

In severe brain injury there may be an elevation of temperature of 106 F. or more. It is a serious mistake to allow the temperature to go higher because of the damage to the brain cells. It may be reduced by bathing with tepid or cool water, by applying ice caps about the body and by an electric fan blowing on the patient's head and front part of the body. The patient should not be covered with bed clothes other than a sheet, and the use of blankets should be condemned.

Rigid Neck

In head injury, especially with the blow being received in the frontal region, there may be a fracture or dislocation of the cervical spine. Roentgenograms of the neck should be included in the examination. A stiff neck, however, is frequently associated with head injury without injury to the vertebra. It may be due to subarachnoid hemorrhage or increased intracranial pressure.

Spinal Puncture

Examination of the spinal fluid is not a routine measure. It should be avoided in the early stages until the patient's condition has been more definitely established, and a localized hematoma has been ruled out. Should the patient, however, be extremely restless with a stiff neck and signs of subarachnoid hemorrhage, a spinal puncture may be of definite therapeutic value. When a spinal puncture is performed, the pressure should be re-

corded because it may give evidence of the degree of intracranial pressure, provided there is no blockage at the foramen magnum or of the ventricular system. Spinal puncture should be contraindicated when there are signs of localized hematoma, as extradural hemorrhage, since the reduction of pressure would increase the formation of the clot and increase the brain compression. A spinal puncture should not be performed in the presence of bleeding or leaking spinal fluid from the ears or nose because of the danger of reversal of the circulation from the contaminated area to the spinal fluid spaces, for fear of producing a meningitis.

Roentgen Examinations

An emergency roentgen examination is seldom indicated in the treatment of injuries of the skull and brain. Should there, however, be a question of a depressed fracture of the skull, it may be made on admission of the patient to the hospital. It may be of aid in diagnosing an extradural hemorrhage when a fracture line crosses the groove of the middle meningeal artery. With these exceptions, an emergency roentgenogram is of doubtful value except to satisfy the curiosity of the physician and of the family. It is useless to make a roentgen examination of a patient who is unconscious and restless when his condition is of such serious nature that it probably will be fatal in a short time. Roentgen examination of a restless, semiconscious patient is of little value because of the poor quality of the picture, and the mere demonstration of a linear fracture would not change the method of treatment. Frequently roentgenograms of the skull are made some days after the injury, depending upon the patient's condition. On roentgen examination, one should look for displacement of the pineal body, because a localized compression from subdural or extradural hemorrhage may displace it to the opposite side.

Treatment

General Care

The emergency room treatment should take care of the immediate needs of the patient and treatment for shock, should it be present. This may require intravenous fluids, plasma or blood transfusion. Lacerations of the scalp may be treated by shaving the head, excision of the ragged, contused edges under local anesthesia and suturing the lesion as an incised wound. If the blood pressure is low and severe shock is present, the repair of the wound should be postponed until a later date. With respiratory difficulty and secretions in the tracheobronchial tree, the patient should be placed in a face down side position with the bed flat, or the foot of the bed elevated to give postural drainage. This position should be maintained as long as the patient remains unconscious and has respiratory secretions in the throat. Should the patient be conscious and more comfortable with an elevation of the head, the back rest may be raised 20 to 30 degrees.

A fluid intake of 2,000 cc. a day is usually adequate, consisting of 1,000 cc. of 5 per cent glucose in normal saline alternating in distilled water every twelve hours by vein. If there are associated injuries of the chest or abdomen or fractured extremities, the fluid intake may be increased to 3,000 cc. a day. It is believed that this amount of fluid intake is adequate to support the patient's general physical needs, and it will not contribute to swelling and edema of the brain. I do not use other dehydrating measures, as solutions of hypertonic glucose, sucrose and magnesium sulfate intravenously, unless there is extreme emergency of relieving intracranial pressure while the operating room is being prepared. I prefer hypertonic sucrose over glucose because of the absence of reversal edema obtained with the latter. Nourishment may be given by nasal tube, with the same amount of fluids of adequate caloric value as mentioned. Intravenous fluids, however, are usually given the first few days with bleeding from the nose and the presence of a considerable amount of mucus in the throat.

Restlessness may be present in a semiconscious patient with a full bladder and with painful areas about the body. Restraints of the body and extremities may increase the restlessness and physical exertion to the point of exhaustion. In order to protect the unconscious patient from falling out of bed and further injuring himself, side boards or rails should always be applied to the bed. Sedation must be used to prevent extreme restlessness and possible exhaustion. In my opinion the best sedative is paraldehyde given by mouth through the nasal tube or by rectum. The barbitals, as phenobarbital sodium given hypodermically, may be used. I prefer not to use opiates unless the patient has associated fractured extremities or severe injury of the chest. In this case I prefer codeine, grains ½, and/or aspirin, grains 10, as needed. Morphine or demerol should be used as a last resort. Opiates have a tendency to depress the respiratory centers, and in severe brain injury

with intracranial pressure the use of them may be fatal.

For a full bladder it is believed that an indwelling plain tip catheter is the best method of treatment. This may relieve the extreme restlessness and keeps the bed dry, aiding in the nursing care of the patient. With the indwelling catheter some form of tidal drainage with continuous bladder irrigation is preferred.

Surgical Treatment

Depressed Fractures of the Skull

Depressed fractures of the skull may be simple or compound. The simple depressed fracture may not require operation as an emergency measure; several days can be allowed to observe the patient's general condition before the operation is performed. Any material depression over important brain centers should be operated on and the depressed fracture elevated to prevent further injury to the brain. Slight depressed fractures over the longitudinal or lateral sinus may be left undisturbed.

Compound depressed fractures of the skull which are frequently comminuted should be operated on as an emergency measure, preferably during the first eight to sixteen hours. Sometimes, with the poor condition of the patient and with adequate chemotherapy, the operation may be postponed twenty-four hours. The indications for the operation are the prevention of infection and the removal of depressed fragments of bone. The blood clots and loose fragments of brain tissue should be removed; then should follow the repair of the dura, which is sutured tightly. The fragments of depressed bone are elevated, and should some of them become detached, they may be replaced in the defect as a graft to fill in the cranial defect. This procedure makes unnecessary at a later date a cranioplasty with the use of a metal plate, which is never as satisfactory as a true bone graft. It is extremely important that the dura be sutured tightly, and should there be a loss of the dura, some temporal muscle fascia or fascia lata may be used as a substitute for the repair. Should the dura not be sutured tightly, the swelling and edema of the brain may force cerebral tissue through the defect and open the scalp wound, producing a brain fungus. This would be a serious complication leading to brain abscess, encephalitis, meningitis and loss of brain tissue, all of which may be fatal. Furthermore, hospitalization would be greatly prolonged because of the delayed healing of the wound.

Extradural Hemorrhage

As soon as the diagnosis of an extradural hemorrhage has been made, an emergency operation should be performed. There is no more extreme emergency in the management of head injuries than that of an acute extradural hemorrhage when a patient may die within a few hours from the time of the injury unless surgical treatment is promptly rendered. At operation the hemorrhage is usually found in the temporal region resulting from the tear of one of the branches of the middle meningeal artery. At times the hemorrhage appears to arise from some of the veins in the neighborhood of the longitudinal or lateral sinus. The operative procedure consists of a drill hole in the temporal region to locate the hemorrhage. Then a small osteoplastic flap is turned down over it for the purpose of removal of the clots and control of the bleeding vessels. After control of the bleeding vessels with electrocoagulation, silver clips, suture or gelfoam, the bone flap is replaced. The advantage of the bone flap is an adequate exposure and the maintenance of the cranial contour after the operation, which is not the case with a subtemporal craniectomy.

Subdural Hematoma

Subdural hematoma may produce the same symptoms and findings as extradural hemorrhage except for the later period of development of several days, and in the case of chronic subdural hematoma it may be weeks or more in coming on. In the case of acute subdural hematoma, usually there is severe contusion and laceration of the brain with unconsciousness from the beginning of the injury, so that the lucid intervals may not be present. With the signs of increased intracranial pressure and a deepening coma, with or without localizing signs, one should consider the possibility of an intracranial hematoma of some type which may require surgical removal. Should there be any doubt of the diagnosis, it is better to make drill holes in both upper temporal regions under local anesthesia to give surgical relief of the hematoma. Sometimes the hematomas are liquid and may be washed out and drained by a catheter, with the use of Ringer's solution. Solid clots may require an enlarged opening or even an osteoplastic flap for their removal. Drill holes may be required in the frontal and occipital regions to aid in the removal when the hematoma extends away from the temporal region. It is better to make drill holes on both sides, especially in chronic subdural

hematoma because of the frequency of the condition on both sides.

Cerebrospinal Rhinorrhea

Leakage of spinal fluid from the ear seldom requires a surgical procedure since it stops of its own accord in a few days. A sterile dressing on the external ear after cleansing with alcohol is all that is required. The external auditory canal should not be plugged up with a packing or with cotton. Chemotherapy with penicillin, sulfadiazine or other antibiotics should be used to lessen the danger of meningitis.

Cerebrospinal rhinorrhea, or the leakage of fluid from the nose, is a more serious complication, and a pyogenic meningitis is apt to develop should the drainage exist over a long period of time. With conservative measures it may stop of its own accord when the opening in the dura is small. To facilitate this stoppage, the patient's head should be elevated on the back rest at an angle of 30 to 45 degrees. The patient should be instructed never to blow his nose, and no sprays nor irrigations in the nasal cavity should be permitted. A spinal puncture should not be performed unless there are definite signs of meningitis, and the leakage of fluid from the nose has stopped, when it may be carried out to determine the type of meningitis.

Should conservative measures fail after one or two weeks, surgical treatment may be used to prevent meningitis. This requires turning down a frontal osteoplastic flap, lifting up the frontal lobe and sewing up the tear in the dura, or repairing the hole with muscle or fascia graft to stop the fluid leakage.

Edema of the brain without hematoma or other form of compression does not require surgical treatment. The old subtemporal decompression for edema of the brain has been discarded. It is doubtful that any surgical measure used today will adequately relieve this form of increased intracranial pressure. Moderate dehydration with limitation of fluid intake, elevation of the head to encourage venous drainage and the maintenance of a free respiratory airway with the administration of oxygen are the principal points in its management.

Convalescent Care

With the regain of consciousness and other evidence of beginning recovery, the head of the patient should be elevated on a back rest preparatory to assuming an erect posture within a week or two. The vasomotor system in the brain, as well as the brain itself, has been injured, and getting the patient up gradually will lessen certain symptoms of vasomotor instability, dizziness, headaches and fainting. It is not necessary to keep the patient in bed for a long period of time. I usually get the patient up within a week or ten days after he becomes symptom-free. With early ambulation the period of convalescence may be shortened and return to employment of light nature will be encouraged.

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The Scientific Department of The Journal

Reflects the Experience of You and Your Colleagues

"Coronary Disease" of the Inner Ear: The Vascular Origin of Vertigo and Deafness

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St. Paul

There is a purpose in drawing an analogy between disorders of the coronary artery and those of the labyrinthine artery of the inner ear. Those who practice in the field of general medicine know a great deal about the coronary artery, its distribution, its disordered function and diseases, and the cardiac disturbances which result. On the other hand, there is too little general knowledge of the same phenomena in the labyrinthine artery and the consequent disturbances in the inner ear. This lack is unfortunate because in clinical medicine vascular inner ear disturbances are at least as common as vascular cardiac disturbances. General ignorance of their meaning denies to these disorders the right of early recognition and treatment. In consequence, in the special field of otology we are too frequently dealing with residual vascular inner ear neural damage in the form of irreversible hearing loss or intractable vertigo, the origin and early phase of which were neglected. In otology we cannot help but feel our guilt. We have not carried a clear message to the general practitioner of medicine about the meaning of tinnitus and early hearing impairment and of initial vertigo, or of the need for their early treatment and of the inevitable end result of neglect in terms of permanent inner ear destruction.

Two important differences between the coronary artery and the labyrinthine artery must be held in mind. Both differences have to do with the end organ they supply:

1. There is a broad spectrum in the work load of the heart and, hence, in its arterial demands on the coronary artery. It varies from the coronary load at complete body rest to the load borne under the most violent body stress. The work load of the cochlear and vestibular end organs of the inner ear and their consequent demands on the labyrinthine artery do not vary significantly in this same sense.

2. The structures of the heart supplied by the coronary artery have none of the functional delicacy which characterizes the neural end organs of hearing and balance in the inner ear. As a result, a vascular change in the cardiac muscle may produce no symptoms while change of equal degree in the labyrinth may be violently expressive in terms of vertigo and/or tinnitus and hearing impairment. By this token the labyrinthine artery may be looked upon as a much more refined indicator of vascular function than is the coronary artery. Exception to this generalization may conceivably be made in the case of the coronary branches to the atrioventricular bundle. Here, in this specialized tissue, one can visualize alteration of conduction and cardiac rhythm in response to similar degrees of vascular change. Excepting those coronary terminals, however, one can appreciate the labyrinth arterial ramifications as more exact indicators of vascular function than comparable coronary branches.

Vascular Dysfunction

Accent is placed in the foregoing statement on vascular function in a broad sense. Vascular function has a pattern and sameness which is system-wide — not restricted to single organs. Differences lie only in the organs supplied. It must be appreciated that vascular disorder, wherever it may occur, is more frequently a factor of vessel dysfunction than of vessel disease. Pathology has dominated medical teaching for so long that present day awareness and accent on physiology and normal and disordered function is thought of as something new in medicine. It, of course, is not. Disease process has always had its first phase in disordered function and its final phase in pathologic change. The inner ear, being graced or cursed with end-circulation without collateral and neural structures of exceeding delicacy, is a clinical paradise for the study of vascular pathophysiology.

There is no pathologic change in the coronary tree that cannot be found in the branches of the labyrinthine artery. The destructive changes in

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the end organs of hearing and balance that result from progressive occlusion of labyrinthine arterial branches are expressed in progressive increase in tinnitus, hearing impairment and vertigo. The changes that result from sudden occlusion, as from thrombus or embolus are cataclysmic in terms of roaring tinnitus, severe deafness and violent vertigo. Studies have shown, however, that inner ear destructions are more frequently due to vascular dysfunction than to these vascular occlusions.

The vascular system in any organ exists that its capillaries can be constantly perfused with circulating blood. The endothelium of the capillary is the exchange barrier between the blood and the dependent tissue. The endothelium is the first tissue cell affected by changes in the blood or alterations in circulation. When this living and semipermeable membrane is damaged, a sequence of misfortune is set in motion — transudate, stasis, hypoxia and peripheral tissue cell starvation. It is this sequence that produces tinnitus and hearing impairment through damage to the cochlear ganglion cells and the neuroepithelium of Corti's organ; and vertigo through damage to the vestibular ganglion cells and the neuroepithelium of the vestibule and semicircular canals. There is a distinctive end vessel distribution in the inner ear. Due to it, certain symptom variations can arise from the end organ. Unfortunately, these variations have been subjected to the common tendency of grouping as syndromes and designation as distinct diseases. Hence, a large variety of symptom complexes have become familiar to you under various headings, as Menière's disease; pseudo-Menière's disease; catarrhal deafness; eighth nerve neuritis; endolymphatic hydrops; labyrinthitis; labyrinthosis, and others. These are not specific and distinct diseases in the true sense. They are symptomatic disorders of vascular dysfunction (in the broad meaning) in an end organ capable of symptom variation due to peculiarities of its anatomy.

Knowledge of vascular dysfunction to a great extent hinges on knowledge of capillary endothelium. The integrity of the endothelium depends upon numerous known and unknown factors. The components of the blood and the adequacy of its circulation through the arteriolar-capillary-venular loop are the factors to which the endothelium is immediately responsive. Once the endothelium is damaged and transudate made easy, characteristics of the extravascular tissues themselves can aid in formation of edema. This is most graphically demonstrated in the retention of extravascular fluid due to unusual concentration of sodium. Its limitation can accordingly have a favorable influence on the undesirable stasis.

The components of the blood known to be important to the endothelium are:

1. The hemoglobin, the principal transport of oxygen vital to the endothelium and to the dependent tissue cells.

2. Certain vitamin catalysts — principally C and probably some B factors.

3. Certain hormones in balanced quantity — principally thyroid and the gonadal secretions.

4. The normal carbohydrate-protein-fat vital to every living cell.

Certain substances in the blood are, with cause, suspected as essential to endothelial function:

1. The adrenocortical hormone now related to the collagen diseases which, in reality, are first transudate diseases.

2. The trace minerals. Their importance is the subject of much current investigation.

The circulation of these elements through the capillary loop is subject to many interruptions. Any halt or delay in their continuous supply to the endothelium can damage the latter and contribute thereby to starvation of the end organ cell. Circulatory interruption may be due to block at the venous or arterial end. In the inner ear venous obstruction is not common. Large and small arterial obstruction due to intimal disease change is, as has been said, as common in the labyrinthine arterial tree as in the coronary. The gradual or sudden starvation of the peripheral capillary endothelium and end organ tissues results in progressive or total inner ear symptoms. Most frequently, however, it is not disease of the arterial walls that causes interruptions to the arterial blood flow, but rather functional obstructions at the arteriolar sphincter level. This sphincter in its widespread distribution through every organ and tissue in the body is the most important smooth muscle in the vascular system. Those factors which cause its sphincteric contractions are the source of arterial obstruction as definite and symptom-producing as atherosclerotic disease and much more common. The factors are principally of two kinds:

1. Direct neural impulsion through autonomic innervation of the arteriolar sphincter. These result from direct central nervous stimulation from the psychic areas most peculiar to man acting through the basal ganglia. Through this direct

neural chain emotional activity can affect arteriolar behavior anywhere in the body. The labyrinthine arterial terminals are commonly thrown into sphincteric spasm by it. The same sequence can derive reflexly from body surface receptors as through chilling or barometric pressure variations.

2. Antigen-antibody influence. Due to previous sensitization of the arteriolar smooth muscle in a so-called shock organ, a state of hypersensitivity is established in it due to antibody association with the muscle. This is prone to happen to the person called "allergic" in the narrow sense of the word. On subsequent re-exposure to the specific antigen, there is antigen-antibody union at the sphincter with resultant injury and spasm.

The end result of these spasmodic interruptions of arteriolar circulation in terms of endothelial damage and end organ stasis, hypoxia and starvation causes, in the inner ear, variations in the symptoms of tinnitus, hearing impairment and vertigo which collectively constitute those many symptom complexes which, by their separation into syndromes and diseases, have brought confusion and poor understanding. I leave it to your imagination to formulate what these same changes at the functional endothelial level in the many other organs and tissues with which you deal produce in terms of symptoms and eventual tissue degeneration, whether in the brain, the joints, the kidneys, the eye, the nose, the intestine, the skin or other organs.

Problem of Diagnosis

It must be apparent, therefore, that the patient who presents the inner ear symptoms of tinnitus, hearing impairment and vertigo in any variation of degree and association should be credited with having an interesting medical problem. Proper differential examination should be directed to the exclusion of occasional intracranial cause due to involvement of central pathways. Since the cochlear and vestibular central pathways diverge, the association of hearing symptoms with vertigo affixes the disturbance in the end organ. The solution of the vascular inner ear problem necessitates an analysis of those factors which can interrupt circulation and of those factors which can primarily cripple the endothelium or a combination of both factors which do commonly co-exist.

Vascular obliterative disease is not uncommon. Vasomotor irregularity in response to physical receptors or emotional reaction is more common. The two not infrequently occur in the same patient. The antigen-antibody conflict of allergy in the narrow sense is only an occasional disturber. The inner ear is not often its shock organ. Inner ear manifestations of endocrine imbalance are commonplace. Hypothyroid states of moderate degree and gonadal problems of premenstrual, pregnant and climacteric nature all can result in easy transudation into the confined inner ear with consequent disability. With increase in our knowledge additional factors will be identified. The particular fundamental problem at hand must be corrected before the essentials of nutrition in a properly nourished person can be made available to the end organ tissues themselves.

Correct treatment is always a factor of etiology. The solution of the vascular disorders of the inner ear and the resultant tinnitus, hearing impairment and vertigo is not a matter of formula but of diagnosis.

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DENVER, COLORADO DECEMBER 2-5, 1952

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From Our President

Citizens First

From his hospital bed a feverish patient surprised his physician by announcing, "I may be a preacher, but I'm a man first, and I can lick you or any of your kin." So with every member of the medical profession; he may be a doctor, but he is a citizen first. In most instances he became a citizen of the United States long before he received the degree of Doctor of Medicine. After he retires from practice, he will still be a citizen. His responsibility to his country preceded his professional obligations and will continue after they cease. As absorbing and demanding as the career of medicine is, it should not preclude the duties of citizenship.

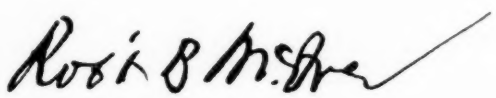
An inherent part of good citizenship is an active, intelligent interest in government. Obviously, the first political obligation of every citizen is to vote. The franchise is the heritage of freedom and is basic to the structure and function of constitutional government. But it must be exercised if freedom is to endure.

Unfortunately, that which is inherited instead of earned is too frequently unappreciated, neglected, treated with indifference, or even abused, squandered and eventually lost. For practical reasons the physician is exempted from jury duty, but no one, not even he, is exempted except in extreme circumstances from the obligation to exercise the franchise.

The physician who fails to engage actively in the affairs of his medical society or to attend hospital staff meetings is in no position to criticize either the medical society or the hospital. Indeed, by failing to make his voice heard he shares the blame for that which is awry. Likewise, the physician who fails to vote is not in good position to complain about taxes, corruption or governmental policies which he finds objectionable. By his surrender of one of the vital duties of citizenship, he assumes a share of the blame for evils at the community, state and national levels.

This election year may well be a significant landmark in American history. Both a privilege and a duty in a nation of free men, the right to vote was never more important than it is now. The fundamental issues of 1952 transcend the usual party politics and affect the future of every American. They call for a clearcut decision by the entire voting population. Let the doctor be sure that he plays his rightful part in that historic decision. It was Edmund Burke who said: "All that is necessary for evil to triumph in the world is for enough good men to do nothing."

To receive the heritage of freedom — first qualify as a legal inheritor. To be a good doctor — first be a good citizen.



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Quo Vadis

The stench of stale tobacco smoke has almost dissipated, the raucous shoutings have settled to a dim reverberation, and another farcical travesty on American democracy has come to an end. Both political parties have nominated their candidates, and the name-calling, claims and counterclaims will now increase in crescendo until election day.

Looking back through history, one finds that few have been the men who actually have changed the world. Certainly, there have been leaders in the past who have been blamed for catastrophic events and others for ushering in new eras of enlightenment. But always, the time was ripe and the people were willing to follow, whether it was Genghis Khan in his attempt to conquer the world or George Washington in his attempt to form a free country. Without the people, these leaders would have been as prophets crying out in the wilderness or crack-pots preaching in Hyde Park on a soap box.

It has been the custom for generations for each political party not in power to bewail the fact that if the regime in control is allowed to continue, the country is bound for bankruptcy or disintegration. It is like a grandfather looking at his progeny mournfully shaking his head, bemoaning the fact that the younger generation is going to pot and will never amount to much. Thus it has been, and thus we hope it will continue to be, but—.

Recent reports from Germany show that denazification programs on the whole have been futile. It is a known psychologic fact that stereotypes formed in childhood persist throughout life, even if their shadings vary a bit. The generation that grew to manhood during Hitler's reign still accepts his teaching as true. It was taught those children by their parents and accentuated in their schools. The roots are deep.

For the past 20 years one political party has been in complete control of our government. This in itself would not be frightening were it not for the fact that for over 12 years a strong personality was at the helm, a man with a purpose, a man on a white horse who in times of despair offered a change — a new deal — in a magnetic, cultured, and compelling voice. The ground was fertile; his seed took root and was fruitful.

To remain static and dormant is to decay. Progress by definition is dynamic. Thomas Jefferson, whom no one would call a communist, believed that changes in government were healthful even if the changes necessitated a small revolution. Certainly in 1932 we all welcomed a change. But with our now gained hindsight, would we again welcome a change from our depression-riddled economy if we knew at the time where this change was to lead us? The "forgotten man" sat on a king's throne. Labor became the power with whom to reckon. Capital was given a nasty connotation. The "pump had to be primed" with staggering government spendings. People joked about the sewers for nowhere the WPA was building, little realizing that it was, after all, their

money that was being poured down those sewers.

What had happened to America? We had had changes in political parties before and hope we may have them again. With the change we obtained in 1932 came a group of high-minded socially conscious gentlemen. They had a purpose. For them to give the citizen a vote and voice in his government was not enough. They, who were the government, must protect this citizen — nurture him from a child with the aid of government monies and help bury him at death with federal funds. If he were unemployed, government monies would pay him. A neat change took place. It is now apparent that the people have stopped running the government — the government is now running them. With the help of wartime emergency powers which continue to exist, the federal government was able to encroach more and more on the inalienable rights of individuals as set forth in democracy's last fort — our Constitution. Always, we were told, it was for the country's good — the individual must yield for the commonweal, — private enterprise was impotent and hence the government must take over this function and that. Indeed private enterprise is becoming impotent solely because government regulations and taxations make any venture for gain or expansion practically impossible. The fetters on industry are so great that only the federal government can engage in new ventures. The incentive of profit is gone. The great blessing of collective bargaining bestowed on the American worker has made him mute before his labor bosses. Yes, this group of high-minded socially conscious political dreamers who took over in 1932 have pretty well proved to us we may never have another depression — neither will we ever have free competitive expansion. We are entering a great new era of mediocrity.

Any bureaucracy seeks to perpetuate itself. Our federal government during the past 20 years has developed more bureaus than all other administrations, the explanation being that more is being done for the people. Evidently what is being done to the people does not matter. Once a bureaucracy reaches a certain size in a democracy, it is impossible to get rid of it because bureaucrats and their relatives control enough votes, and do vote, to win any election.

In the coming election there are numerous issues at variance in the two parties, but basically only two are of importance. Firstly, do we wish to perpetuate further the present bureaucracy and dynasty filled as it is with graft, dishonesty, and lack of true leadership? Secondly, do we wish to continue the paternalistic government set up in 1932, which can and will eventually lead to a new type of American government which may be democratic in name but certainly socialistic in action? Do we wish to relinquish our right to free endeavor, involving as it does the prospect of lean years as well as prosperous ones, for a lifetime of mediocre living?

Basically, the Democratic Party's campaign will be built around the philosophy that capital, labor and the farmer "never had it so good." If they are giving us socialism, then it is socialism we need and want. Certainly on the surface this is an excellent argument — one which won Harry S. Truman the presidency in 1948 — but an argument which is not true according to the Democrats' own Department of Labor statistics. These show that dollar for dollar, the average worker has less dollar buying ability than in 1939.

The Republican Party offers us a promise of reform — a crusade to sweep out from office those who by their lust for power, greed and corruption crave more and more authority to secure their bureaucracy more firmly. It offers also a hope to engage in free endeavor without the threat of the government taxing the incentive out of industry or crucifying a profession on the altar of a welfare state.

The very fact that we still have elections shows that American Democracy is not dead. It will only die if you fail to vote. It is not believed that either candidate of the two major political parties is of such strength and calibre that he alone will change the course of our history. The continuation of our present policies, however, will eventually lead to a point of no return. The seeds sown and cultivated for 20 years have taken root. Some may never be uprooted. Maybe none should. The choice is yours, to continue onward or change course. Quo Vadis?

Civil Defense Medical Supplies

Of all federal money spent on civil defense, approximately 70 per cent is going for medical purposes, chiefly matching grants to states for local medical stockpiling and all-federal regional stockpiles.[1] The annual report of Administrator Millard Caldwell of the Federal Civil Defense Administration, covering the first full year's operations, reveals that half of some $100,000,000 ap-

propriated for all purposes has been earmarked for all-federal medical purposes. Twenty million will be expended either for federal medical purchases or matching grants to states.

The report estimated that by June 1952 about $90,000,000 in medical supplies would be procured or would be on order. The extra $20,000,000 was to come from nonfederal sources.

During 1951, regional, state and local civil defense directors received specifications for about 200 individual medical items, which they were privileged to order independently or through the Federal Civil Defense Administration. It was expected that within the present year 58 regional warehouses would become available for stockpiling of federal supplies, both medical and engineering.

State and local officials are being encouraged to employ the "unitizing" system used in the federal warehouses. To be immediately available at the time of emergency, supplies are warehoused by the "unitizing" technic. Instead of all items of a given type, such as surgical instruments, being stored in one area, items forming a unit are stored together. At the time the report was issued, plans called for storage of medical supplies and equipment in units necessary for a 200 bed emergency hospital. States were being urged to store their own supplies in units sufficient to care for 1,000 casualties. In an emergency, a local community would have to depend on its own medical supplies pending arrival of medical units from the nearest federal regional warehouse.

It is planned that on the federal level there will be exchange of perishable medical supplies with other government agencies. States expect to rotate their own perishable items with state hospitals and other institutions.

For the fiscal year 1953, the program calls for appropriations from the Congress amounting to $193,000,000 for federal medical stockpiling.[2] No matching funds for local stockpiling are requested for next year.

1. 70% of Federal Civil Defense Funds Go for Medical Purposes, Capitol Clinic 3:2 (April 29) 1952.
2. Federal, State Medical Stockpiles May Exceed $80 Million by Mid-Year, Capitol Clinic 3:3 (April 1) 1952.

Quiet for Heart Patient

Make haste slowly in moving a patient immediately after a heart attack. Dr. Elmer Milch and his associates of Buffalo, N. Y., gave this advice at the recent meeting of the American Medical Association. It was prompted by investigations which led them to conclude that perhaps two thirds of the sudden deaths from cardiac disease which occur in young men are preventable if the patient is not moved during the crucial hours right after the attack and is kept very quiet.

They advise keeping the patient resting quietly at home, on in his office if he suffers the attack there, for the first four hours, using morphine or other sedatives to keep him quiet and giving oxygen if necessary. They stress not moving him too soon, for such a patient, when rushed to the hospital, is too often dead on arrival or dies shortly thereafter.

In their animal experimentation these investigators observed that in dogs which were anesthetized and prepared for study of the effects of blocking of the coronary arteries, the condition often terminating fatally in young men, fibrillation and death followed the slightest outside stimulus, such as brushing against the operating table. In their efforts to reduce these effects of outside stimuli, they prevented the stimuli from reaching the heart by cutting the sympathetic nerves in the chest and thereby reduced the death rate from 42 to 14 per cent. They considered this accomplishment good evidence of the importance of keeping the patient absolutely quiet during the crucial period of several hours immediately following the attack.

Reports on Poisoning Sought

A nationwide toxicologic study of cases of poisoning resulting from the use of insecticides, rodenticides, fungicides, weed killers, fumigants, repellents and related types of chemicals used in agriculture and the home currently is being undertaken by the Committee on Pesticides of the Council on Pharmacy and Chemistry of the American Medical Association. The information obtained will be used to expand the committee's permanent file of such cases for use by physicians and allied medical personnel.

The committee is appealing to physicians to submit records on cases of nonfatal and fatal poisonings from pesticides. It is functioning as a center for reporting poisoning cases of this type, and unpublished isolated cases coming to its attention have supplied much of the information it has compiled. In most instances, summary data on the pertinent facts of the poisoning and the circumstances of their occurrence furnish sufficient information.

This appeal should evoke wide interest. Doubtless Florida physicians, particularly in the citrus areas, will have cases to contribute to the committee's file.

ILO — Danger Ahead!

To American physicians today, the three initials "ILO" spell danger ahead. Or at least they should, for they stand for an international body which eventually may change the whole concept of medical practice throughout the world.

Surprising as it may seem, socialized medicine the international way is now the favored approach. The time has come when under United Nations plans an attempt is being made through treaty-making powers to dictate the relationship between American citizens and their government. It is an astounding fact that this great nation, vaunted as leader of the free world, is now under greater threat of socialism by treaty than by domestic legislation. The immediate source of that danger is the ILO.

The International Labor Organization is an affiliated agency of the United Nations comprising some 60 active member-governments. It is one of the oldest and most important of the maze of international organizations functioning today. Its policy-making body is the International Labor Conference, which meets annually, usually in Geneva, Switzerland, where the staff maintains permanent headquarters.

At its annual meeting last June, this conference exercised its authority of formulating international treaties in its adoption of the so-called "Social Security Minimum Standards Convention of 1952." This convention (treaty) recommends that member nations put into effect minimum standards of social security in nine fields — medical care, sickness benefits, unemployment benefits, old age pensions, employment injury benefits, family allowances, maternity benefits, invalid benefits, and survivors' benefits. Carefully distributed throughout the covenant, the medical benefits plainly constitute socialized medicine when considered together.

The medical care section, for example, stipulates in part that a country ratifying must provide a system of compulsory health insurance, or (1) private, voluntary health insurance administered by public authorities under established regulations set by law, or (2) private, voluntary health insurance administered by insurance companies under government supervision.

Now that the domestic battle against political medicine has been won, at least for the time being, in state legislatures and the Congress, the new international approach threatens loss of the war against this evil, largely because of lack of recognition of the possibility of political and economic regimentation from external sources. With the public unaware of the dangers inherent in the treaty-making power, the nation's international representatives have jeopardized national sovereignty and the Constitution as well as American medicine by "wholesale approval of treaties, conventions, and executive agreements."

Ratification of this newest ILO convention requires only a two-third vote of the Senate. Such a vote would establish socialized medicine in this country regardless of the wishes of the majority of the members of the Congress and the public to the contrary. That it could slip in through the backdoor under the guise of an international treaty seems all but inconceivable, but this subtle approach to realization of the socialist's dream of cradle-to-the-grave security is the reason why ILO spells danger.

The time is surely ripe for physicians to acquaint themselves fully with this latest development and to realize that only the opening skirmish in the socialized medicine battle may be behind them. The American Medical Association has for years kept watch of the ILO and lately has published a number of articles on this organization and its health objectives. This material[1] is regarded as "must" reading for every physician. Do not miss it, Doctor.

1. For back reading, physicians are invited to turn to the Journal of the American Medical Association: Organization Section, May 31, 1952; page 869 of the June 28 issue, containing the excellent statement of A.M.A. President Louis H. Bauer; the Washington News Section of the issue of July 12, 1952; the editorial on page 1572 and the Organization Section of the issue of Aug. 23, 1952; and Capitol Clinic Vol. 3, No. 26, July 1, 1952.

Southeastern States Cancer Seminar Tampa, Oct. 30 - Nov. 1

The Southeastern States Cancer Seminar will be held this year at the Tampa Terrace Hotel in Tampa. The dates for this sixth annual meeting are October 30 and 31 and November 1. The attendance during the last five years has been excellent, and it is anticipated that physicians throughout the Southeastern states will welcome the exceptional opportunity again this year and attend in large numbers.

The American Cancer Society, Florida Division, and the Florida State Board of Health, with the cooperation of the Florida Medical Association, sponsor this annual Seminar.

The lecturers and the specialty they represent are: Chemotherapy, Dr. C. P. Rhoads, Memorial Center, New York City; Hematology, Dr. Leon

Jacobson, University of Chicago Clinic, Chicago; Gynecology, Dr. John L. McKelvey, Head of Obstetrics and Gynecology, University of Minnesota Medical School, Minneapolis; Lip, Mouth, Throat, Dr. John J. Conley, Pack Medical Group, New York City; Pathology, Dr. Hugh G. Grady, Director, American Registry of Pathology, Armed Forces Institute of Pathology, Washington, D. C.; Pathology, Dr. Elson B. Helwig, Armed Forces Institute of Pathology, Washington, D. C.; Radiology, Dr. Charles L. Martin, Dallas; Radiology, Dr. W. Edward Chamberlain, Philadelphia; Skin, Dr. Joseph A. Elliott, Jr., Charlotte, N. C.; and Surgery, Dr. Clarence Gardner, Duke University Hospital, Durham, N. C.

Postgraduate Assembly in Endocrinology Miami Beach, November 3-8

The fifth annual Postgraduate Assembly in Endocrinology and Metabolism will take place on Nov. 3-8, 1952 at the Roney-Plaza Hotel in Miami Beach. This assembly is sponsored by the Endocrine Society, formerly the Association for the Study of Internal Secretions, and is being held in Miami Beach for the second time in two and one-half years. Registration is limited to 200 and closes October 1. The fee is $75.

An intensive and practical review of the newer developments in endocrinology and metabolism will be presented, with emphasis on the relation of these subjects to general medicine. The program has been accepted for credit by The American Academy of General Practice.

A faculty of 20 eminently qualified clinicians and investigators will present 64 scientific lectures covering all phases of endocrinology and metabolic disorders. There will be case presentations of various types of endocrine and related problems and also question and answer periods. Laboratory procedures of aid in endocrine diagnosis will be demonstrated by trained technicians. In addition, scientific exhibits will be presented by members of the faculty, and a bound copy of the proceedings will be available to registrants.

This is one of the most important postgraduate seminars held in this country and is the first, at least in this area, in which simultaneous translations into Spanish of all presentations will be provided through earphones. Wide publicity has been given this seminar in the medical press of Latin American countries, and several governments have already announced their intention of sending official medical representatives. Those in attendance from these countries have been extended an invitation to attend the annual meeting of the Southern Medical Association, which will be held in Miami the following week, November 10-13.

Dr. Carlos P. Lamar, Director of the Department of Endocrinology and Metabolism of the Jackson Memorial Hospital in Miami, is a member of the distinguished faculty and is also chairman of the local committee arranging for the meeting. Dr. Henry H. Turner, Secretary-Treasurer, The Endocrine Society, 1200 N. Walker, Oklahoma City 3, Okla., is receiving applications.

Southern Medical Association Meeting Miami, November 10-13

The forty-sixth annual meeting of the Southern Medical Association will convene in Miami on Monday, Nov. 10, 1952 and will continue through Thursday, November 13. The Dade County Medical Association will serve as host to this body for the fourth time and is making elaborate plans for welcoming the physicians of the South to Florida's popular convention city.

In contrast to arrangements for the last meeting held in Miami, all activities will be centered in the downtown bayfront area, in hotels on Biscayne Boulevard and in the Library and Municipal Auditorium in Bayfront Park, thereby eliminating the transportation problem.

An excellent program has been arranged by the officers of the twenty-one sections. There are Sections on General Practice, Medicine, Gastroenterology, Neurology and Psychiatry, Pediatrics, Pathology, Radiology, Dermatology and Syphilology, Allergy, Physical Medicine and Rehabilitation, Industrial Medicine and Surgery, Surgery, Orthopedic and Traumatic Surgery, Gynecology, Obstetrics, Urology, Proctology, Ophthalmology and Otolaryngology, Anesthesiology, Public Health, and Medical Education and Hospital Training. The diversity of subjects to be presented and the distinguished essayists insure broad coverage of all aspects of medical progress.

President R. J. Wilkinson of Huntington, W. Va., who was unable to attend the Dallas meeting last year because of illness, is actively engaged in meeting the customary demands of his high office in preparation for this meeting. He will be succeeded in office at the close of the meeting by Dr. Walter C. Jones of Miami, a past president of the Florida Medical Association.

Medical District Meetings October 27-31, 1952

Dr. Eugene G. Peek, Jr., of Ocala, Chairman of Council, has announced that the programs for the four annual Medical District Meetings have been completed. These programs, scheduled for October 27, 29, 30 and 31, have been arranged by Dr. Peek with the assistance of the eight councilors. Cooperating in the many details of the meetings are the secretaries of the host county medical societies: Bay, Dr. William F. Humphreys, Jr., of Panama City; Pinellas, Dr. Whitman C. McConnell of St. Petersburg; St. Lucie-Okeechobee-Martin, Dr. Adrian M. Sample of Fort Pierce; and Volusia, Dr. Robert L. Miller of Daytona Beach.

Medical District Meetings are designed to enable Association officers and members to meet together with a minimum of traveling for those attending, and so that busy doctors will be away from their practices as short a time as possible. The scientific program of each meeting has been planned to be sufficiently diversified to be of value to general and specializing practitioner alike.

Following the scientific program, officers of the Association will present pertinent and interesting information at each of the meetings. Association officers who will speak include Drs. Robert B. McIver, president; Frederick K. Herpel, president-elect; Samuel M. Day, secretary-treasurer; and Shaler Richardson, editor of The Journal. Dr. J. Rocher Chappell, chairman of the Committee on Advisory to Selective Service for Physicians and Allied Specialists, will join with the officers in bringing timely information to the members. These general sessions will be in charge of Dr. Peek, assisted by a councilor from each district as follows: Panama City, Dr. Benjamin A. Wilkinson of Tallahassee; St. Petersburg, Dr. Hugh G. Reaves of Sarasota; Fort Pierce, Dr. Donald W. Smith of Miami; and Daytona Beach, Dr. Eugene L. Jewett of Orlando.

All sessions begin at 2:30 p.m. on the days indicated on the program below. At 5:45 refreshments will be served by the host societies. Dinner will follow at 6:30. Printed programs will be mailed to all members of the Association prior to the meetings.

Dr. H. Phillip Hampton, Chairman of the Committee on Legislation and Public Policy, will preside at the evening session, beginning at 7:30. Legislation on state and national levels of interest to the medical profession will be the subject for discussion. Association members and members of the Woman's Auxiliary are urged to attend.

Scientific Assemblies — Four Medical Districts

All Meetings — 2:30 p.m.

Monday, October 27, 1952

Panama City — A

St. Andrews Bay Yacht Club

Presiding: Eugene G. Peek, Jr., Chairman of Council, and Francis M. Watson of Marianna, Councilor of District 1.

Address of Welcome, Charles H. Daffin of Panama City, President, Bay County Medical Society.

"A Phase of Postpartum Bleeding with Case Reports," Merritt R. Clements, Tallahassee.

"Thoracic Problems of Interest to the General Practitioner," (by invitation), Hawley H. Seiler, Tampa.

Wednesday, October 29, 1952

St. Petersburg — C

Detroit Hotel

Presiding: Eugene G. Peek, Jr., and Emmett E. Martin of Haines City, Councilor of District 6.

Address of Welcome, John P. Rowell of St. Petersburg, President, Pinellas County Medical Society.

"Management of Thoracic Problems in General Practice," Leffie M. Carlton, Jr., Tampa.

"The Female Urethra in the Diagnosis of Abdominal and Pelvic Pain," Melvin M. Simmons, Sarasota.

Thursday, October 30, 1952

Fort Pierce — D

Flamingo Restaurant

Presiding: Eugene G. Peek, Jr., and Erasmus B. Hardee of Vero Beach, Councilor of District 7.

Address of Welcome, Richard F. Sinnott of Fort Pierce, President of St. Lucie-Okeechobee-Martin County Medical Society.

"Diabetic Neuropathy," Sidney Davidson, Lake Worth.

"The Early Diagnosis and Management of the Common Neoplasms," Richard M. Fleming, Miami.

Friday, October 31, 1952

Daytona Beach — B

Daytona Beach Gun Club

Presiding: Eugene G. Peek, Jr., and William C. Thomas, Jr., of Gainesville, Councilor of District 3.

Address of Welcome, J. Richard West of Daytona Beach, President, Volusia County Medical Society.

"Problems in Sterility," Edward P. Madden, Daytona Beach.

"Management of the Ambulatory Cardiac," Norman E. Williams, Daytona Beach.

Dr. Bauer, A.M.A. President, Addresses Florida Medical Committee For Better Government

An address by Dr. Louis H. Bauer, President of the American Medical Association, highlighted the final session of the third annual meeting of the Florida Medical Committee for Better Government. Following a luncheon in the George Washington Hotel Auditorium at 12:30 p.m. on Sept. 7, 1952, Dr. Bauer spoke on "Problems of Medicine, National and International" before approximately 500 Florida doctors and their wives. He was introduced by Dr. Robert B. McIver of Jacksonville, President of the Florida Medical Association.

Dr. Bauer opened his address by saying, "The American Medical Association having thrown itself into the struggle to protect the freedom of medicine, much of its publicity has been devoted to the matter of socialized medicine. Now, however, that we have won a breathing space in our struggle, we are endeavoring to bring to the attention of the public and the profession the other matters with which we are concerned and which, as a matter of fact, have always occupied the major portion of our efforts. The entire environment of medical practice today is due to these activities of the American Medical Association. The very fact that every medical school in the United States is an acceptable school is the direct result of the Association's campaign initiated shortly after the turn of the century to raise the standards of medical education in this country. As a result, not only do we have nothing but high grade medical schools, but the level of medical education here is unsurpassed anywhere in the world.

"The work of the councils and bureaus of the American Medical Association," continued Dr. Bauer, "has set standards in hospitals and in medical care which likewise are unsurpassed. The profession and the public have been protected against undesirable secrecy in objectionable advertising of proprietary medicines. Standards have been set for therapeutic agents, special purpose foods, devices and methods employed in the practice of medicine. Quackery, nostrums and charlatanism have been shown up and in many cases eliminated. The Association has done much and is expanding its program to educate the public in matters of health through radio and television projects, its program on school health and its consultation service to governmental and lay organizations. Its publications are outstanding and cover the field of medicine. The Journal of the American Medical Association is recognized as the greatest medical publication in the world."

After enumerating the many services rendered by the various councils and bureaus, Dr. Bauer commented, "I find in going about the country that many members of the Association and many members of the general public are totally unaware of these activities, or if they are aware of them, they take them for granted.

"History has shown that whenever groups within the medical profession start to bargain independently, the influence of the profession is lost. That has been the case in Great Britain. If one had been asked 25 years ago what medicine of the future would be like, I doubt if anyone could have come up with a very revealing answer. I am

not sure that anyone can do so in any very certain fashion for the next 25 years. Certain patterns, however, have developed which at least indicate definite trends. Some of these trends are heartening, particularly those in medical science. Other trends, particularly those in the socioeconomic field, are not only disheartening but are rather alarming. If some of these latter trends are allowed to continue unchecked, the very science of medicine will suffer."

Observing that there is no reason to believe any disease exists for which a cure and prevention will not be found eventually, Dr. Bauer continued, "All this leads to longer life. The average life span has increased 20 years in the last 50 years and will probably continue to increase. This brings up economic problems. At the present time, the increased life span has resulted in millions living to an age where they are subject to the degenerative diseases accompanying old age. This interposes an immediate problem, the care and treatment of chronic and degenerative diseases.

"In the last quarter century the status of the physician is altered in his relations to society and government, greatly in some areas, and to a certain extent in all areas. Government has come to play an increasing part in medicine. To some degree this was essential. Many phases of public health cannot be adequately controlled without the participation of government. For example, environmental sanitation, protection of food, milk and water supplies, and control of insect-borne diseases must be a function of government health departments, but such matters must be carried out on a community basis and require a police power."

This distinguished servant of medicine was firm in his conviction that the medical profession should lead in any health program. Commenting on health plans, he said, "The prepayment system of medical care is here to stay. The question to be decided is whether the system should be voluntary or compulsory. In many areas of the world, it is compulsory. In this country, so far, we have kept it on a voluntary basis. Whether or not we continue to do so depends largely on the medical profession. Many seem to think that we have won our fight against socialized medicine. I assure you we have not and we cannot relax. We have won a breathing space. The socializers are not for the moment trying to move in a program of socialized medicine through the front door, but they are still trying to get it in by degrees through the back door. They have not given up, nor will they give up until the American people show that they will have no more of socialism whether it is in the form of admitted socialism or in the form of pink sugar-coated socialistic medical pills.

"I have had the opportunity during the last five years to visit Europe ten times. In not one single country have the plans involving greater participation of government resulted in a betterment of medical care, and in many they have resulted in a poorer type of medical care. The medical profession is usually at odds with the government. The government ignores the medical profession, and professional matters are decided by laymen. There seems no doubt that wherever government has established a strangle hold on medical care, it is the eventual aim of government to make doctors full time salaried servants of the state. History shows that wherever government gets its foot in the door of medical care, there is more and more governmental control, more and more regimentation, steadily increasing deficits in the insurance plan and deterioration of the standards of medical care. The public is the loser.

"In this country, medical care has become political. The majority of the hopeful proponents of compulsory insurance are either socialistic in their thinking, or are using the proposal as a lure to get votes by offering the public something they cannot possibly deliver. The Health Minister of Australia has stated in substance that it is impossible to make any health plan work which will not enlist the cooperation of those who have to carry it out. In other words, no health plan can be carried out without the help of the medical profession, and, if they disapprove the plan, it cannot be made a success."

Dr. Bauer outlined the steps necessary to prevent socialized medicine in this country. "We must see that the scope of public health units as defined by the American Medical Association is generally accepted, and then coverage within that scope must be extended to the whole country. Diagnostic facilities must be made available to the whole country. Diagnostic facilities must be made available within the reach of any community. Hospital facilities must be adequate, but full use must be made of existing facilities before new ones are built. Local pride or local politics must not be allowed to result in the building of a hospital in an area which does not have one when adequate facilities are available within a few miles and good roads lead to those facilities. Agreements must be made between the medical profession and

local government agencies to insure adequate medical care to the indigent.

"There are now 86 million people in the United States who carry hospital insurance. The medical profession must give 100 per cent cooperation to the voluntary plan. The doctors themselves must clean house. Those physicians who are charging exorbitant fees are constantly getting the medical profession into discredit with the public. Whereas only a small proportion are guilty of so doing, those few do more harm than ten times the number of ethical physicians can make up for because they are the ones who get the publicity.

"Health and medicine require teamwork, but the physician must be captain of the team. The medical profession must stand together as a unit. The confidential relationship between physician and patient must be maintained. If that is lost, medicine will degenerate into an assembly line product. When it is said that government is the only agency with enough money to give financial support to medical schools, my answer is that the government has no money except that which it takes out of your pocket or mine. Our federal government has expanded into such an octopus that it reaches into every corner of our lives. It has been extravagant and wasteful. It has seized nearly all available sources of taxation, leaving little to the state. Should the government gain control of our medical schools by furnishing the greater part of their financial support, do not let anyone persuade you that the government would never interfere with the operation of the schools.

"The Federal Security Agency is the greatest propaganda agency for socialism in the United States today. It has not been content with trying to force a program of socialized medicine on the people of the United States, but has gone at least into one other country and tried to force such a program on the people of that country. Our free institutions and traditions are in grave danger of being lost by the apathy of the American people. In the last presidential election only 51 per cent of those eligible to vote exercised their right of franchise. That means that 26 per cent of the people control the country. Only in a few places in the world does this precious right of franchise remain. Centuries ago Plato said the penalty paid by good men for refusing to take part in government is to live under the government by bad men.

"Our form of government has been steadily changing from a democracy within a republic to a bureaucracy. The people of this country have been encouraged to depend upon government for everything in life. Once personal liberties are lost, they are never regained. We have now reached the stage where nearly 35 per cent of our income goes to national, state and local taxation. This is a dangerous level. Beyond that point, complete socialism becomes inevitable. The most important crusade this or any organization can make is a crusade for good citizenship and acceptance of civic responsibilities. Use your influence to get people to register and vote, regardless of how they vote.

"Finally, we must arouse the American people to a realization that if medical service is to progress, if the standards of medical education and medical care are to be maintained and advanced, then socialization not only of medicine but of anything else in our national economy cannot be tolerated, or we shall lose everything that has made the United States of America the greatest country in the world."

Dr. Bauer's address climaxed a two day gathering of the Florida Medical Committee for Better Government. Senator J. Marshall Butler (R.Md.) spoke to the group at the opening luncheon at the George Washington Hotel at 1 p.m. on Saturday, September 6. At 8:30 that evening Congressman Charles E. Bennett (D.Fla.) was the speaker. The public was invited to hear both political speakers. Their addresses were carried on the radio, as was Dr. Bauer's address.

The agenda for Sunday included a business meeting at 9:30 a.m. Newly elected officers are: Chairman, Dr. Samuel G. Hibbs, Tampa; Vice Chairman, Dr. Francis T. Holland, Tallahassee; Secretary, Dr. Collin F. Baker, Jr., Tampa; Treasurer, Dr. Charles F. Henley, Jacksonville. The District Chairmen are: District 1, Dr. Francis H. Langley, St. Petersburg; District 2, Dr. O. E. Harrell, Jacksonville; District 3, Dr. James T. Cook, Jr., Marianna; District 4, Dr. Richard F. Stover, Miami; District 5, Dr. C. Robert DeArmas, Daytona Beach; District 6, Dr. Alvin E. Murphy, Palm Beach; District 7, Dr. Jere W. Annis, Lakeland; District 8, Dr. J. Maxey Dell, Jr., Gainesville.

At this business session, the Florida Medical Committee for Better Government, which functions apart from any organized county, state or national medical society, went on record as endorsing Gen. Dwight D. Eisenhower for president, Sen. Richard M. Nixon for Vice President, Sen. Spessard Holland for re-election as the senior

senator from Florida and Dan McCarty for governor of Florida. It was emphasized at the time of these endorsements that the Committee as such would not carry on any active campaign, but it recommended to its individual members that they support the candidates as endorsed.

STATE BOARD OF HEALTH

Treatment of Selected Cancer Patients At Oak Ridge Institute of Nuclear Studies

Arrangements have been made whereby selected cancer patients from Florida may be treated at the Oak Ridge Institute of Nuclear Studies at Oak Ridge, Tenn. The State Board of Health Cancer Division Director has been appointed consultant by the Oak Ridge Institute to act as clearing house for those patients from Florida who may be treated at this Institute.

There is no charge for hospitalization at the Institute, regardless of the patient's finances. However, transportation must be paid by the patient or some other source from his home to Oak Ridge and return. A booklet giving information to the patients has been made available to each of the tumor clinics in the state and each patient considered for admission should be familiar with the contents of this booklet. Before any patient is sent to Oak Ridge application has to be made on a special form and approved by the Director of the Cancer Division of the State Board of Health. The hospital at Oak Ridge is a very small hospital and no patient should be referred there without proper credentials. All patients should be processed through one of the fourteen tumor clinics in the state if possible.

The Institute is interested in treating only the following types of patients:

1. Patients with bone metastases, particularly osteogenic in type, who have already had the benefit of standard forms of therapy.
2. Osteogenic sarcoma in any stage of the disease.
3. Carcinoma of the thyroid.
4. Patients with pleural effusions or ascites caused by metastatic neoplasms and who have prominent symptoms related to the presence of the fluid. Patients who are becoming rapidly worse or who have impending intestinal obstruction are not suitable.
5. Patients with plasma cell myeloma. The Institute would especially like to study 1 or 2 patients with plasma cell leukemia.
6. Chronic granulocytic leukemia.
7. Polycythemia vera.
8. Acute leukemia in adults with high peripheral white cell counts.

The hospital is also interested in having:

1. Any histologic sections for their files, on cases referred.
2. A brief clinical summary on all cases.
3. In patients with ascites on pleural effusions they would like to know the amounts of paracenteses.
4. Any x-rays that have been made on the patients will be helpful and these will be photographed and returned if desired.

It is believed that this is a service that will be helpful to the types of cancer cases mentioned above. If there are any questions about this service inquiries may be forwarded to Dr. L. L. Parks, Cancer Division, State Board of Health, Jacksonville.

BIRTHS, MARRIAGES AND DEATHS

Births

Dr. and Mrs. Richard W. McDowell of Jacksonville announce the birth of a son, Jeffrey Jules, on July 6, 1952.

Dr. and Mrs. John S. Cowdery of Jacksonville announce the birth of a son, Robert Collins, on July 26, 1952.

Dr. and Mrs. Thaddeus M. Moseley, III, of Jacksonville announce the birth of a son, William Armistead, on Aug. 11, 1952.

Dr. and Mrs. Jerome H. Newman of Jacksonville announce the birth of a daughter, Fanny, on Sept. 7, 1952.

Dr. and Mrs. Irvin C. Schneider of Jacksonville announce the birth of a son, Kenneth Evans, on Sept. 7, 1952.

Marriages

Dr. David S. Light of Miami Beach and Miss Letty Pogul of Forest Hills, N. Y., were married on Aug. 30 1952.

Deaths — Members

Sperry, Herbert G., Coral Gables ..... July 6, 1952
Owens, W. Duncan, Warrington, Va. ..... Aug. 6, 1952
Pay, Wilson C., DeLand ..... Aug. 12, 1952
Harrison, M. M., Bradenton ..... Aug. 16, 1952
Ricker, Samuel F., Orlando ..... Aug. 18, 1952
Weems, William H., West Palm Beach ..... Aug. 30, 1952

Deaths — Other Doctors

Thompson, Hubert O., Tyler, Tex. ..... March 7, 1952
Vogt, Elkin, Lithonia, Ga. ..... May 5, 1952
Pernoud, Flavius G., Jr., St. Louis, Mo. ..... June 9, 1952

STATE NEWS ITEMS

Distribution of the American Medical Association publication, "Your Money's Worth in Health," is being carried out in Sanford by Dr. J. Clifford Boyce in cooperation with a local druggist who has agreed to wrap one of the booklets with each prescription. Dr. Boyce said the plan assures that the publication will go to the person for whom it was originally intended. The druggist says he is going to do more than wrap the booklet with each prescription. He's going to ask the customer to read it.

Dr. Douglas D. Murphy of Venice spoke on "Prolonging Life" at a recent meeting of the local Rotary Club.

Dr. Webster Merritt of Jacksonville was the principal speaker at a dinner held during the refresher course for pharmacists in Jacksonville in June. His subject was "A Down to Earth Talk."

Dr. J. Archer Smith, Jr., of Homestead was honored recently for a quarter-century of service at Dade County Hospital. More than 100 friends witnessed the presentation to Dr. Smith of a scroll signed by all the doctors and personnel of the hospital.

Dr. Sullivan G. Bedell of Jacksonville has been elected vice president of the Board of Directors of the Duval County Child Guidance Clinic. He has served as president for the past year.

Dr. Clarence M. Sharp of Jacksonville has been elected to the Executive Council of the American Trudeau Society.

Dr. Samuel E. Kaplan of Venice has returned to his practice after atending a postgraduate course in August.

Drs. Hugh A. Carithers and Luther W. Holloway of Jacksonville gave lectures at the Southern Pediatric Seminar held in Saluda, N. C., in July.

Dr. Turner Z. Cason of Jacksonville was moderator for a discussion on the need for a program of professional education in heart diseases before a meeting of the Duval County Chapter of the Florida Heart Association in June.

Dr. Wiley T. Simpson of Winter Haven is attending the meeting of the World Medical Association in Athens, Greece, October 12 through 16.

Dr. J. Champneys Taylor of Jacksonville has returned to his practice after attending clinics and conferences at the Mayo Clinic.

Dr. C. Burling Roesch of Jacksonville was the principal speaker at the commencement exercises of the second graduating class to receive practical nursing certificates at the Jacksonville School of Technology.

Dr. W. Tracy Haverfield of Miami spoke on brain surgery at the second session of the Institute on Nursing, sponsored by the University of Miami and the Florida League of Nursing Education, district two, recently. Dr. DeWitt C. Daughtry of Miami spoke on surgery of the heart.

Dr. Percy H. Guinand of Clearwater attended the International Diabetic Congress in Amsterdam, Holland during July.

Dr. Homer L. Pearson, Jr., Miami, was a guest speaker at the joint convention of the New Hampshire Medical Society and the Vermont State Medical Society, at Brettonwood, N. H., on September 9.

Representatives from Florida at the Medical Public Relations Institute at the Edgewater Beach Hotel in Chicago, September 4 and 5, included Ernest Gibson and Tom Jarvis from the Association office and Mrs. Louis Johnson from the Dade County Medical Association.

Sponsored by the Department of Public Relations of the American Medical Association, this working clinic was designed primarily for lay personnel of state and county medical societies.

WANTED — FOR SALE

Advertising rates for this column are $5.00 per insertion for ads of 25 words or less. Add 20c for each additional word.

FOR SALE: Spencer Microscope; binocular; inclined lens mounting; mechanical stage; practically new; perfect condition; case included. Write: JDM, 1708 Conway, Orlando, Fla.

WANTED: M.D. for general practice in Mulberry, Fla. Address all inquiries to the City Council, Mulberry, Fla.

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NEW MEMBERS

The following doctors have joined the State Association through their respective county medical societies.

Jiminez, Manuel E., Key West

Kaplan, Sherman R., Miami Beach

Kicklighter, James E., Key West

COMPONENT SOCIETY NOTES

Dade

The Dade County Medical Association was recently presented with a citation by the Miami Chamber of Commerce for the work of its members in encouraging employers to hire the physically handicapped. The presentation was made on the program, "Hope Unlimited," over Station WQAM. Physicians are guest speakers on the weekly program which is sponsored by the Chamber of Commerce.

At the September 3 meeting of the Dade County Medical Association, a panel discussion on facial pain was held. Taking part were Drs. Philip Weinstein, Thomas Cook, Kenneth S. Whitmer and Christian Keedy.

Escambia

Escambia county physicians were lauded by *The Pensacola News* in an editorial, Aug. 13, 1952. The newspaper stated that Escambia county surgeons performed 148 operations at Escambia General Hospital during 1951 without charge for their services. "Surgeons take turns in performing these operations, thus carrying on the tradition of the profession that no person in need of medical attention shall suffer because of financial distress." Further it was pointed out: "Too often the unthinking are prone to criticize the medical profession and hospitals for their high fees and charges. They do not stop to figure how costs have risen and what the profesison and the hospitals do to see that medical services and hospitalization is denied no one because of his inability to pay."

Lake

The Lake County Medical Society has paid 100 per cent of state dues for 1952.

Leon-Gadsden-Liberty-Wakulla-Jefferson

The Leon-Gadsden-Liberty-Wakulla-Jefferson County Medical Society Medical Society will sponsor the Florida Medical Association's Fair Exhibit at the North Florida Fair in Tallahassee, Oct. 27-Nov. 1, 1952. Dr. Francis T. Holland, chairman of the Society's committee on public relations, and Dr. George H. Garmany, both of Tallahassee, are in charge.

Palm Beach

All members of the Palm Beach County Medical Society have paid their state dues for 1952.

Pinellas

The meeting of the Pinellas County Medical Society held on September 5 was the first following the customary recess during the summer months.

The Pinellas County Medical Society is now publishing "The Aesculapian," a reception room supplement to "The Picomeso Mail Bag," the Society's official publication. This section is handy to remove and place in reception rooms to be read by patients.

Seminole

The Seminole County Medical Society's radio program on station WTRR, Sanford, was three years old the last of August, according to announcement by Dr. Orville L. Barks, chairman of the Society's committee on public relations. Transcriptions from the record library of the FMA Bureau of Public Relations have been used for the program.

OBITUARIES

James Covington Richardson

Dr. James Covington Richardson of Miami died on May 25, 1952. He was 58 years of age.

Dr. Richardson was born in 1894. He received his medical training in Nashville, Tenn., and in 1920 was awarded the degree of Doctor of Medicine by the Vanderbilt University School of Medicine. His specialty was surgery. He had practiced in Miami since he came there from Monroe, N. C., in 1932. Locally, he was a member of the Rod and Reel Club and a former member of Harvey Seeds Post of the American Legion. He retained membership in the Baptist Church of Monroe.

A member of the Dade County Medical Association, Dr. Richardson was also a member of the Florida Medical Association for 20 years and held membership in the American Medical Association.

Surviving are the widow, Mrs. Minnie Brooks Richardson; and three sons, James C. Richardson, Jr., Charles H. Richardson and Alfred B. Richardson.

William Benjamin Hopkins

Dr. William Benjamin Hopkins died of a heart attack at his home in Tampa on April 27, 1952. He was 68 years of age.

Born in Franklin County, Virginia, on Dec. 8, 1883, Dr. Hopkins was the first son of William Leftwich Turner and Mary Ella Hopkins. He attended the local schools, the Virginia Polytechnic Institute, and the Medical College of Virginia, from which he was graduated in 1907. He also studied at the University of Vienna in Austria, and while engaged in postgraduate work there served as president of the American Medical Association of Vienna.

Upon entry of the United States into World War I, he enlisted in the Army Medical Corps and from July 1918 to April 1919 served with the American Expeditionary Forces. He was discharged from military service with the rank of major in 1919.

Dr. Hopkins specialized in ophthalmology and otolaryngology. He practiced in Richmond, Va., until 1926, when he moved to Tampa and continued the practice of his specialty there for a quarter of a century. Active in civic and fraternal affairs, Dr. Hopkins was a past commander of USS Tampa Post No. 5, American Legion, and a member and former president of the American Legion Round Table of Hillsborough County. Other affiliations included Sons of Confederate Veterans, Sons of the American Revolution, Society of Colonial Wars, Military Order of the World War, the Stars and Bars, a Military Order, Elks, Knights of Pythias, Hillsborough County Association for the Blind and the Tampa Lions Club, in which he was awarded a life membership for his work in its clinics for the blind. He held membership in the Theta Nu Epsilon and Omega Upsilon Phi fraternities and was also a member of Ye Mystic Krewe of Gasparilla. Active in the Hyde Park Methodist Church, he had served as a member and as chairman of its board of stewards.

Dr. Hopkins was a member of the Hillsborough County Medical Association, the Florida Medical Association and the American Medical Association.

Surviving are the widow, the former Mary C. Nicholson of Littleton, N. C.; one son, Dr. William B. Hopkins, Jr., a lieutenant in the Navy Medical Corps; one daughter, Mrs. Charles S. Heinmiller of Rochester, N. Y.; four brothers, O. L. Hopkins of Richmond, A. H. Hopkins of Rocky Mount, Va., Dr. C. D. Hopkins of Tampa, and Lawson M. Hopkins of Peoria, Ill.; and two sisters, Mrs. D. V. Sabine of Yonkers, N. Y., and Miss Anne Hopkins of Rocky Mount, Va. Three grandchildren also survive.

Leo Henry Riesenbeck

Dr. Leo Henry Riesenbeck of Miami died on June 12, 1952 after being in failing health for some years. He was 48 years of age.

A native of Ohio, Dr. Riesenbeck was born in Cincinnati on Aug. 12, 1903. He received his early education there and was a graduate of Miami University. In 1932, the degree of Doctor of Medicine was conferred upon him by the University of Cincinnati College of Medicine.

Until 1939, Dr. Riesenbeck engaged in the general practice of medicine in Norwood, Ohio. He then spent three years in Barnes Hospital in St. Louis and in Cincinnati General Hospital. In 1942 he was certified by the American Board of Otolaryngology.

After successfully practicing his specialty in Cincinnati for five years, he came to Florida in 1947 because of failing health. After two years his health had improved so much that he was able to enter the general practice of medicine in Miami, and he anticipated resuming his specialty. Within a year, however, after his retirement in 1951 this seasoned clinician, experienced specialist and skillful surgeon lost his battle against disease.

Dr. Riesenbeck was a member of the Dade County Medical Association. He became a member of the Florida Medical Association in 1950 and for the last two years had held honorary status. He also was a member of the American Medical Association.

Adrian Colonna Coogler

Dr. Adrian Colonna Coogler died at his home in Brooksville on June 18, 1952 after a short illness. He was 67 years of age.

The son of Col. and Mrs. Theodore S. Coogler, who came to Brooksville from Manning, S. C., in the eighteen-seventies, Dr. Coogler was born in Brooksville in 1884. He attended local schools and received his medical education in Virginia. In 1907, he was awarded the degree of Doctor of Medicine by the Medical College of Virginia at Richmond.

Returning to his birthplace to reside, Dr. Coogler engaged in the general practice of medicine in Hernando County for 45 years. At the time of his death he was County Physician. He was a member of the Egypt Temple Shrine, of Tampa, the Woodmen of the World, and the Methodist Church.

Dr. Coogler was a member of the Pasco-Hernando-Citrus County Medical Society and had for 35 years been a member of the Florida Medical Association. He was also a member of the American Medical Association.

Surviving are the widow, Mrs. Christine Coogler; one son, Theodore Coogler of Brooksville; one daughter, Mrs. Evelyn C. Kilby of Gainesville; two sisters, Mrs. B. C. Floyd of Brooksville and Mrs. F. I. Roberson of Green Cove Springs; two brothers, F. B. Coogler and J. K. Coogler, both of Brooksville; and one grandson, John Adrian Kilby of Gainesville.

Arthur Jones Logie

Dr. Arthur Jones Logie of Miami died suddenly on June 13, 1950. He was 44 years of age.

Born in New York City in 1907, Dr. Logie pursued his education at Columbia University and the University of Tennessee, and also in Scotland. He attended the Royal Academy of Edinburgh where he was graduated in medicine in 1933, and also received a degree from the University of Glasgow. He interned in the Edinburgh Royal Infirmary and later in the United Hospital at Port Chester, N. Y. After a residency in the Sea View Hospital in New York City, he became affiliated with the Florida State Hospital at Chattahoochie.

Thereafter, he did research work on the BCG vaccine for the New York City Department of Laboratories at the Willard Parker Hospital until

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"Migraine may appear during the first years of life. The presence of subjective signs, such as headache and flimmer scotoma, is often difficult to determine in young children. The true nature of the symptoms frequently remains obscure for years."

Vahlquist, B. and Hackzell, G.: Acta Paediatrica *38*: 622 (1949).

NO. OF CASES	SEX	AGE AT ONSET	CYCLIC VOMITING	DURATION OF ATTACK	INTENSITY
31	8 ♀ 23 ♂	3 yrs. (mean)	3 out of 31	2½ hrs.	severe in all cases

TABLE CONT'D

NO. OF CASES	UNILATERAL HEADACHE	NAUSEA	FLIMMER SCOTOMA	VERTIGO	HEREDITY
31	18 out of 31	31 out of 31	12 out of 31	6 out of 31	20 out of 31

(reference given above)

In a study of 400 adult migraine patients, it was revealed that 34% had suffered attacks before the age of 15.* These investigators concluded that childhood migraine was a much greater clinical problem than was previously believed and that psychodynamic mechanisms played an important part in the disease.

These criteria are useful in diagnosis:

Headache attacks with symptom-free intervals plus (at least two of the following) nausea, scintillating scotoma, hemicrania, and hereditary predisposition.

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In the adolescent age group, 12 to 18 years of age, the dosage may gradually be increased as necessary up to the usual adult dose, i.e., two tablets when the attack appears imminent followed by one tablet doses at half hour intervals until the attack is aborted. *(Total maximum dose for adults: six tablets for each attack.)*

*Katz, J., Friedman, A.P., and Gisolfi, A.: New York State J. Med. *50*: 2269 (Oct.) 1950.

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1936. At that time he came to Jacksonville to become medical director of the Tuberculosis Division of the Florida State Board of Health, which he organized, and he served in this capacity until 1941. Here he instigated the first mass screening of the people of the state for the detection of tuberculosis, a project which has become nationwide.

Dr. Logie entered the private practice of medicine in Miami in 1941. Already a lieutenant in the United States Army Reserve since 1934, he expected his call to active duty, which, owing to a chronic thrombophlebitis, never came. He did, however, secure active duty as a surgeon in the United States Public Health Reserve, in which he became a captain and served from 1941 to 1950. Locally, he spent much of his time in the cardiac section of Jackson Memorial Hospital and he was active in furthering the development of the Miami Heart Institute, of which he became a director in 1950. He was a Scottish Rite Mason and a Shriner, and enjoyed membership in the Mahi Shrine band.

A member of the Dade County Medical Association, Dr. Logie was also a member of the Florida Medical Association and the American Medical Association. He had been a fellow of the American College of Physicians since 1940 and of the American College of Chest Physicians since 1950 and a member of the American Trudeau Society since 1937. He also held membership in the National Tuberculosis Association and was a diplomate of the American Board of Internal Medicine.

Surviving are the widow, Mrs. Marie Ford Logie, and three sons, Arthur J., Jr., Andrew John and Michael Bruce, all of Miami, and the parents, Mr. and Mrs. Leo L. Logie, and a brother and a sister, all of California.

George Walton Carter

Dr. George Walton Carter of Chipley died on May 5, 1952 at his office following a heart attack. He was 60 years of age.

A native of Georgia, Dr. Carter was born at Lake Park in 1891. He received the degree of Doctor of Medicine from the Maryland College of Eclectic Medicine and Surgery in 1913.

Dr. Carter served in World War I and was honorably discharged with the rank of captain. In 1917, he settled in Caryville, where he served a

physician for two lumber mills. An ardent baseball fan, he organized and managed the Caryville Lumberjacks, a well known area baseball team of several years ago. Since 1937 he had practiced medicine in Chipley. He was a Mason and a Shriner, and a member of the Chipley Methodist Church.

This widely known Washington County physician was serving as president of the Washington-Holmes County Medical Society at the time of his death. For 26 years he had held membership in the Florida Medical Association and he was also a member of the American Medical Association.

Survivors are the widow, Mrs. Maude Davis Carter of Chipley; two foster daughters, Mrs. Mike Toole of Graceville and Mrs. Alex H. Lewis of Marianna; four sisters, Mrs. Senora Thomas of Plant City, Mrs. Mary Hinely and Mrs. Lucy Brown, both of Howell, Ga., and Mrs. James Turner of Savannah, Ga.; and three brothers, Lewis Carter and Paul Carter, both of Lake Park, Ga., and Haywood Carter of Alva.

Medical Licenses Granted

Dr. Homer L. Pearson, Jr., Secretary of the State Board of Medical Examiners, has reported that of the 247 applicants who took the examinations of the Board, held June 30 and July 1, 1952, in Jacksonville, 220 passed and have been issued licenses to practice medicine in Florida. The names and addresses of the 220 successful applicants follow:

Anderson, Katherine Hunter, Winston-Salem, N. C. (Cornell U. 1940)
Anderson, Oliver David, Grand Rapids, Minn. (Coll. of Med. Evangelists 1950)
Andrews, James William, Miami (U. of Maryland 1952)
Andrus, Don Leverett, Pensacola (George Washington U. 1943)
Armbruster, Robert Homer, Springfield, Ill. (Northwestern U. 1951)
Artler, Vernon Benson, East Ann Arbor, Mich. (Temple U. 1949)
Augustus, Charles Anderson, (Col.), Muskogee, Okla. (Howard U. 1949)

Bacon, Alfons R., Chicago, Ill. (U. of Illinois 1921)
Bailey, Taylor Dodge, Kissimmee (U. of Buffalo 1951)
Baker, J. Allen, Chicago, Ill. (U. of Illinois 1951)
Balthrop, John Edward, Jr., Pensacola (Med. Coll. of Alabama 1951)
Barker, Arthur Joseph, Pensacola (U. of Vermont 1948)
Barnes, Zerney Burns, Jr., Jacksonville (Emory U. 1950)
Barrineau, Charles Edward, Cairo, Ga. (Emory U. 1952)
Beeson, James Dennis, Jacksonville (U. of Indiana 1949)
Bennett, David Dresden, Jr., St. Augustine (Northwestern U. 1946)

Advertisement

From where I sit
by Joe Marsh

Wonder how Miss Gilbert Is in "Histery"?

By now I guess you've heard about the spelling errors in the kids' report cards this week.

A typical card looked like this:

Arithmetic.....B
Geography.....B–
Spelling.....C
Grammer.....B

I don't know if Miss Gilbert, the principal, actually wrote those cards, but she took full responsibility. This morning she got up in the Assembly Hall — before the students — and started writing GRAMMAR with two "a's" on the blackboard 100 times!

From where I sit, I'll bet this makes her even more popular with the students. It's nice to see an expert admit she can make a mistake. Too many so-called "experts" claim they're never wrong on such subjects as what you or I ought to eat . . . how we should practice our profession . . . whether we should enjoy beer or buttermilk. A really wise person never claims to "know all the answers."

Joe Marsh

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Bernstein, Theodore Clark, Levittown, L. I., N. Y. (New York U. 1945)
Biggane, Charles Francis, Jr., Miami (U. of Pennsylvania 1945)
Bilotta, Laurence Alexander, Orlando A. F. Base (St. Louis U. 1935)
Birdsong, Gordon Gilbert, Brewster (U. of Tennessee 1939)
Bissell, Lewis Frederick, Jacksonville (U. of Arkansas 1949)
Blumenfeld, Irving Herman, Brooklyn, N. Y. (Chicago Med. Sch. 1939)
Boucek, Robert Joseph, Miami Beach (U. of Pittsburgh 1943)
Bradley, James Lawrence, Altoona, Pa. (Yale U. 1943)
Bradley, Matthew Henry, Warrington (Ohio State U. 1951)
Brill, Warren Daniel, Silver Spring, Md. (U. of Maryland 1944)
Burley, Lee, Richmond, Va. (U. of Louisville 1947)
Byrd, William McCulloh, West Palm Beach (Emory U. 1952)

Cady, Donald James, Saginaw, Mich. (U. of Michigan 1948)
Cantor, Leonard Carroll, Miami (Med. Coll. of Virginia 1952)
Carpenter, Samuel Louis, Jacksonville (Temple U. 1947)
Carter, John Paul, Ann Arbor, Mich. (George Washington U. 1945)
Chambers, William Norman, East St. Louis, Ill. (Washington U. 1950)
Choisser, John Elder, DeLand (St. Louis U. 1939)
Collins, Thomas Rodney, Orlando (U. of Chicago 1940)
Conrad, Elmer R., Detroit, Mich. (U. of Minnesota 1932)
Cox, Forest William, Dayton, O. (U. of Pennsylvania 1925)
Coy, Francis Matthew, Bay Pines (U. of Louisville 1942)
Cummings, Eugene Heyward, Jacksonville (Columbia U. 1944)

Daniel, Ben Early, Lake Butler (U. of Georgia 1945)
Davis, David Kern, Miami (Georgetown U. 1948)
Davis, John Llewellyn, St. Petersburg (Vanderbilt U. 1932)
Dell, George Alexander, Jr., Gainesville (St. Louis U. 1952)
Deutsch, Irvin, Coral Gables (Dalhousie U. 1938)
Dormon, George, Winter Haven (Med. Coll. of Alabama 1950)
Drake, Robert Eldon, Winter Park (Louisiana State U. 1948)

Eccles, Claude Graham, Staten Island, N. Y. (Tulane U. 1932)
Edwards, Ralph, Miami (Boston U. 1952)
Ehlert, Albert John, Lakeland (Tulane U. 1945)
Elgin, Lee William, Jr., Miami Beach (U. of Maryland 1952)
Emerson, Richard Putnam, Boston, Mass. (Harvard Med. Sch. 1952)

Farrar, Edward Larston, Jr., Charleston, S. C. (Emory U. 1949)
Felder, Louis Homer, Atlanta, Ga. (Emory U. 1946)
Fishbein, Harry, Houston, Tex. (U. of Arkansas 1938)
Fisher, John Edward, New Castle, Ind. (U. of Pennsylvania 1943)
Fletcher, John Clyde, Greensboro (Emory U. 1952)
Flinchum, Darius, Warm Springs, Ga. (U. of Virginia 1944)
Fontaine, William C., Panama City (U. of Buffalo 1943)
Frazier, Calvin Hunter, Huntington, W. Va. (Med. Coll. of Virginia 1951)
Frell, Joseph Calabria, Miami Springs (Ohio State U. 1937)
Friedl, Lois Evelyn, Pensacola (U. of Illinois 1950)

Galison, Louis, Miami (Long Island Coll. of Med. 1931)
Gallagher, James Francis, Jr., Newtonville, Mass. (Tufts Coll. 1947)
Garby, Rodes Carl, St. Petersburg (George Washington U. 1943)

Garrett, Spencer Roberts, Columbus, Ga. (Duke U. 1952)
Gattas, Fred Albert, Chicago, Ill. (U. of Illinois 1945)
Gayden, John Matthew, Orlando (Med. Coll. State of S.C. 1951)
Geiger, Eric Francis, Lake Butler (Tulane U. 1952)
Germain, William McKee, Lantana (U. of Michigan 1921)
Gilbert, Robert Gordon, Tampa (Emory U. 1952)
Glassman, Jacob Abraham, Chicago, Ill. (U. of Illinois 1935)
Golinvaux, C. J., Monroe, Mich. (St. Louis U. 1921)
Graves, Herschel Anderson, Jacksonville (Vanderbilt U. 1948)
Gryte, Lewis Adolph, Queen's Village, N. Y. (Manitoba Med. Coll. 1921)
Guerin, Briant Bowman, Sumter, S. C. (Duke U. 1941)
Guest, Maurice Calvin, Orlando (Coll. of Med. Evangelists 1946)
Gunther, Roy W., Amsterdam, N. Y. (Columbia U. 1931)

Habegger, Myron L., Rockledge (U. of Indiana 1932)
Halpryn, Hillard Jack, New York, N. Y. (Columbia U. 1952)
Harrow, Benedict Richard, Pittsfield, Mass. (Duke U. 1945)
Hartman, Clifford Duane, St. Petersburg (Loyola Sch. Med. 1936)
Hastings, Edward Val, Milwaukee, Wis. (Marquette U. 1944)
Hastings, Lawrence Vaeth, Miami (Johns Hopkins Med. Sch. 1948)
Hatfield, Jack Joseph, Indianapolis, Ind. (Indiana U. 1943)
Herr, Paul Sylvester, Harrisburg, Pa. (Hahnemann Med. Coll.of Phila. 1933)
Hobach, George Bernard, Johnson City, Tenn. (U. of Pennsylvania 1943)
Hobach, John Philip, Johnson City, Tenn. (U. of Pennsylvania 1943)
Hornsby, Aubrey Thomas, Durham, N. C. (Columbia U. 1946)
Howard, Karen, Washington, D. C. (Vienna, 1938)
Huntington, Frederic Sargent, Darien, Conn. (Harvard Med. Sch. 1924)
Huth, Edgar William, Valders, Wis. (U. of Wisconsin 1931)

Jackson, Truxton Lawrence, South Miami (Vanderbilt U. 1940)
Jaffe, Norman, Ft. Campbell, Ky. (New York U. 1946)
Jennings, Erwin Reeves, Milledgeville, Ga. (U. of Maryland 1946)
Jeruss, Edward George, White Plains, N. Y. (U. of Arkansas 1940)
Johnson, Whedon, Baltimore, Md. (Johns Hopkins Med. Sch. 1943)
Jones, Elizabeth Charlotte, Branford (Tulane U. 1952)
Jones, Samuel Thomas, III, Birmingham, Ala. (Tulane U. 1950)

Kartsonis, Apostolos Athos, Starke (Athens, 1937)
Kearns, Walter Melody, Jr., Chicago, Ill. (Johns Hopkins Med. Sch. 1947)
Keese, Harvey, Jr., Oak Ridge, Tenn. (U. of Tennessee 1951)
Kindler, Jack, Miami Beach (Duke U. 1952)

Lahser, Charles Irving, Roseboro, N. C. (Bowman Gray Sch. of Med. 1946)
Lane, Martin Luther, Humboldt, Tenn. (U. of Tennessee 1950)
Langston, Henry Irwin, Marianna (Tulane U. 1946)
Langston, Richard James, Atlanta, Ga. (Emory U. 1952)
LaRose, William Robert, Tampa (Loyola U. 1952)
Lauth, Edward John, Jr., Cp. LeJeune, N. C. (U. of Pittsburgh 1947)
Lavengood, Russell W., Jr., New York, N. Y. (U. of Louisville 1951)
Lay, Coy Lafayette, Lakeland (U. of Texas 1946)
Lazzopina, Michael Salvatore, Jersey City, N. J. (Georgetown Med. Sch. 1948)

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Lee, John Portmess, St. Petersburg (Washington U. 1941)
Leffall, LaSalle Doheny, Jr., (Col.), Quincy (Howard U. 1952)
Leonard, James Bernard, Clearwater (Jefferson Med. Coll. 1944)
Lerman, Philip Harry, Westbury, N. Y. (U. of Maryland 1944)
Lerner, Lee Wolfe, New York, N. Y. (McGill U. 1919)
Lev, Morris, Miami Beach (Creighton U. 1934)
Lucas, John Richard, Lake City (Western Reserve U. 1941)
Lundell, Edward Sigfrid, Jacksonville (U. of Pittsburgh 1951)
Lyons, Carmine Keith, Durham, N. C. (Duke U. 1946)

Mack, Daniel Elliott, Jacksonville (Syracuse U. 1946)
Mathews, James Richardson, Jacksonville (U. of Cincinnati 1950)
McCanless, James Thomas, Independence, La. (Med. Coll. of Georgia 1952)
McCarthy, Joseph Francis, Jacksonville (Hahnemann Med. Coll. of Phila. 1948)
McClanathan, Donald Eugene, Chicago, Ill. (U. of Illinois 1944)
McCreary, Albert Bordelocque, Jacksonville (U. of Tennessee 1952)
McLaren, John Robert, Jacksonville (Temple U. 1948)
McLeod, Wallace Norman, Atlanta, Ga. (Emory U. 1952)
Medlock, Frederick Eugene, Jr., Decatur, Ga. (Emory U. 1951)
Mendelblatt, David Lee, St. Petersburg (George Washington U. 1927)
Messer, Henry Davis, Madison (Duke U. 1950)
Meyer, George Wright, Jacksonville (Duke U. 1952)
Miller, Neill Davis, Louisville, Ky. (U. of Louisville 1948)
Miller, William Winfred, Jr., Warrington (U. of Wisconsin 1943)
Millikan, William Johnson, Indianapolis, Ind. (Indiana U. 1942)
Mohler, Floyd William, St. Petersburg (Indiana U. 1944)
Monroe, William Daniel, Pensacola (Tulane U. 1950)
Montague, Fairfax Eubank, Durham, N. C. (Emory U. 1952)
Moore, Maurice Roberts, Norwich, Conn. (Queens U. 1929)
Morgan, Bernard Laurence Newlands, Jacksonville (London, 1940)
Morgan, Paul William, Brackenridge, Pa. (U. of Pittsburgh 1944)
Morris, Richard William, Ft. Lauderdale (U. of Vermont 1930)
Mosher, Donald Melvin, Century (Med. Coll. of Alabama 1949)
Mudd, Robert Hiden, Miami (U. of Pennsylvania 1951)
Muir, Ian Wildridge, Birmingham, Ala. (Med. Coll of Alabama 1951)

Neill, John Storts, Inverness (U. of Louisville 1946)
Nelson, John Hall, Birmingham, Ala. (Med. Coll. of Alabama 1950)
Newman, Abe Lowell, Miami (Med. Coll. of Virginia 1951)
Newman, Jerry Irving, Canton, O. (Ohio State U. 1946)
Norby, Richard Gerhard, New Orleans, La. (U. of Minnesota 1946)

Oleson, Dunlap Wrisley, Hinsdale, Ill. (U. of Chicago 1946)
Osterman, Floyd Arthur, Lake City (Northwestern U. 1945)

Packer, Marvin S., Miami Beach (Chicago Med. Sch. 1950)
Parks, Charles Thomas, Warrington (Med. Coll. of Georgia 1951)
Parmley, John Bill, Electra, Tex. (Tulane U. 1946)
Patterson, Charles Archley, Warrington (Tulane U. 1942)
Perkins, William Hale, Jesup, Ga. (Harvard Med. Sch. 1945)
Pfister, Howard Frederick Carl, Cincinnati, O. (U. of Cincinnati 1943)
Porter, Clark Gardner, Three Rivers, Mich. (Washington U. of St. Louis 1935)

Ralyea, John, Paw Paw, Mich. (Wayne U. 1943)
Ramel, William Joseph, Miami Beach (Jefferson Med. Coll. 1945)
Read, John Sturdivant, Brooklyn, N. Y. (Long Island Coll. of Med. 1902)
Reece, John Wesley, Collbran, Ala. (New York U. 1946)
Rhodes, William Minner, Columbus, O. (U. of Cincinnati 1941)
Rickerd, Charles Lloyd, Milton (Northwestern U. 1941)
Robelen, Harry Charles, Jr., Triangle, Va. (Med. Coll. of Virginia 1942)
Rollings, Harry Evan, Savannah, Ga. (Tulane U. 1943)
Romoser, William Carl, Columbus, O. (Ohio State U. 1935)
Rosenbaum, Irving Henry, Lake City (Columbia U. 1934)
Rubenstein, Eli, Cincinnati, O. (U. of Cincinnati 1946)
Rumberger, Wilson Allen, Pittsburgh, Pa. (U. of Pittsburgh 1950)
Sall, Walter Gerald, Miami Beach (New York U. 1952)
Schadel, Lees Malcolm, Jr., Ft. Lauderdale (Hahnemann Med. Coll. of Phila. 1941)
Scheribel, Karl, Ft. Lauderdale (Loyola Med. Sch. 1931)
Schimek, Robert Alfred, Baltimore, Md. (Johns Hopkins Med. Sch. 1950)

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Schlapik, Daniel David, Miami Beach (Northwestern U. 1925)
Seaman, Irvin, Oak Park, Ill. (Chicago Med. Sch. 1945)
Shedd, Robert Harry, Warrington (U. of Pennsylvania 1948)
Shmerling, Abram Carl, Nashville, Tenn. (Vanderbilt U. 1952)
Silver, Abraham, Miami Beach (St. George Med. Coll. 1936)
Simon, Howard M., Jr., Cleveland, O. (U. of Pittsburgh 1951)
Simpson, Forbes Rheaume, Maitland (Temple U. 1952)
Sinatra, Thomas Joseph, Brooklyn, N. Y. (New York U. 1952)
Slagle, David James, Tampa (U. of Tennessee 1951)
Smathers, Charles Ralph, Philadelphia, Pa. (Temple U. 1952)
Smith, William Landon, Henderson, Ky. (U. of Louisville 1949)
Spindler, James Francis, Dunedin (U. of Georgia 1936)
Stewart, Thomas Jones, Century (Med. Coll. of Alabama 1947)
Stuart, Jack Frederic, Coral Gables (Cornell U. 1951)
Sublett, Daniel Voorhees, Lexington, Ky. (U. of Louisville 1927)
Talcott, LeRoy Everett, Jr., Cp. LeJeune, N. C. (Duke U. 1947)
Thompson, James Ellsworth, Chattahoochee (Emory U. 1952)
Thompson, Ramon Converse, Decatur, Ga. (Emory U. 1952)
Thompson, Ronald MacKinnon, West Palm Beach (U. of Chicago 1949)
Thomson, Robert Vail, Coral Gables (U. of Iowa 1942)
Trappolini, Alma, Miami (U. of Tennessee 1949)
Trumbo, Robert Benson, Orlando (Emory U. 1952)
Turner, Oliver Edmonds, Pittsburgh, Pa. (Jefferson Med. Coll. 1936)
Tyor, Malcolm Paul, McRae, Ga. (Duke U. 1946)
Underwood, Charles R., St. Louis, Mo. (Emory U. 1952)
Utkov, Edmund, Memphis, Tenn. (Switzerland 1947)
Vallotton, William Wise, Valdosta, Ga. (Med. Coll. of Georgia 1952)
Vaughan, Edgar Allen, Jr., Tampa (Emory U. 1952)
Vaughn, Arthur Raymond, Jr., Philadelphia, Pa. (Jefferson Med. Coll. 1947)
Virusky, Edmund Joseph, Jesup, Ga. (U. of Maryland 1941)
Wager, William Francis, Bainbridge, Ga. (Emory U. 1951)
Waldron, Barzillia Robbins, Brookline, Mass. (New York Med. Coll. 1945)
Walker, Gilbert Harold, Key West (Tulane U. 1952)
Walker, Harry Leigh, Gainesville (Temple U. 1952)
Weber, Henry Charles, Drexel Hill, Pa. (Marquette U. 1914)
Weisberg, Abraham Allen, Detroit, Mich. (Detroit Coll. of M. & S. 1932)
Welebir, Andrew John, Falls Church, Va. (Coll. of Med. Evangelists 1939)
Wessel, George Henry, V, Miami (Temple U. 1952)
Whitaker, Jo Anne, Winter Haven (Bowman Gray Sch. of Med. 1952)
White, Gilbert H., Jr., Lynch, Ky. (U. of Illinois 1951)
Wiswell, Orville Otto, Miami (St. Louis U. 1952)
Whitten, Samuel Biscow, Jacksonville (Tulane U. 1952)
Wolf, Earl George, Pensacola (U. of Illinois 1941)
Young, Corbin Alfred, New Orleans, La. (Tulane U. 1952)
Zavertnik, Joseph James, Coral Gables (Johns Hopkins Med. Sch. 1943)

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SURGERY—Intensive Course in Surgical Technic, Two Weeks, starting October 6, October 20, November 3. Surgical Technic, Surgical Anatomy & Clinical Surgery, Four Weeks, starting October 20. Surgical Anatomy & Clinical Surgery, Two Weeks, starting November 3. Surgery of Colon & Rectum, One Week, starting October 20, November 17. Gallbladder Surgery, Ten Hours, starting October 20. Bronchoscopy, One Week, by appointment. General Surgery, One Week, starting October 6. General Surgery, Two Weeks, starting October 6. Breast & Thyroid Surgery, One Week, starting October 6. Esophageal Surgery, One Week, starting October 13. Thoracic Surgery, One Week, starting October 20. Fractures & Traumatic Surgery, Two Weeks, starting October 6.

GYNECOLOGY—Intensive Course, Two Weeks, starting October 20. Vaginal Approch to Pelvic Surgery, One Week, starting November 3.

OBSTETRICS—Intensive Course, Two Weeks, starting September 29, November 3.

MEDICINE—Intensive General Course, Two Weeks, starting October 13. Gastroenterology, Two Weeks, starting October 27. Gastroscopy & Gastroenterology, Two Weeks, starting November 3.

CYSTOSCOPY—Ten-Day Practical Course starting every two weeks.

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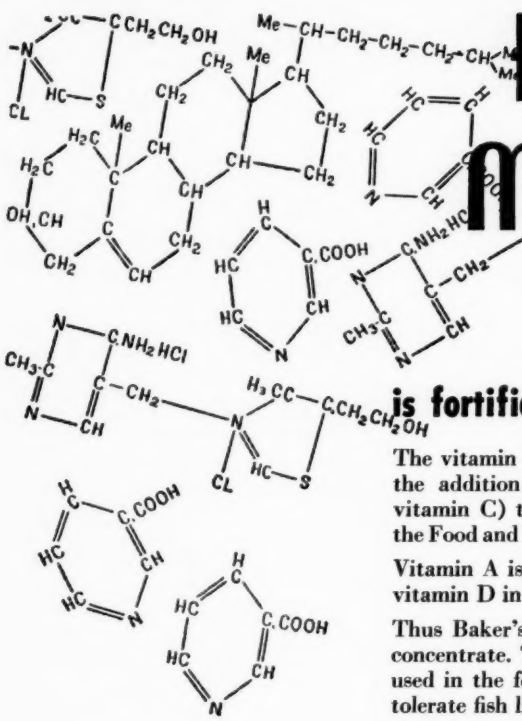

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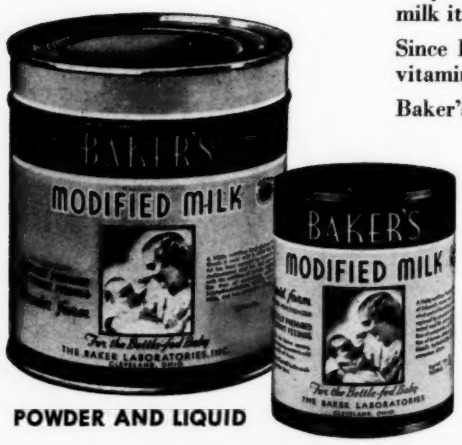

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Riboflavin	1.0 Milligrams

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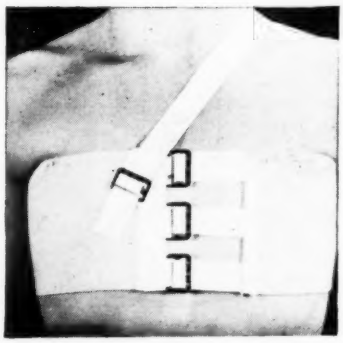
SCHEDULE OF MEETINGS

ORGANIZATION	PRESIDENT	SECRETARY	ANNUAL MEETING
Florida Medical Association	Robert B. McIver, Jacksonville	Samuel M. Day, Jacksonville	Hollywood, Apr. 26-29, '53
Florida Medical Districts	Eugene G. Peek, Jr., Ocala	Council Chairman	
A-Northwest	Francis M. Watson, Marianna	Benjamin A. Wilkinson, Tallahassee	Panama City, Oct. 27, '52
B-Northeast	William C. Thomas, Jr., Gainesville	Eugene L. Jewett, Orlando	Daytona Beach, Oct. 31, '52
C-Southwest	Emmett E. Martin, Haines City	Hugh G. Reaves, Sarasota	St. Petersburg, Oct. 29, '52
D-Southeast	Erasmus B. Hardee, Vero Beach	Donald W. Smith, Miami	Ft. Pierce, Oct. 30, '52
Florida Specialty Societies			
Academy of General Practice	Edward F. Shaver, Tampa	Frank T. Linz, Tampa	Hollywood, Apr. 26, '53
Allergy Society	Nelson Zivitz, Miami Beach	Solomon D. Klotz, Orlando	" "
Anesthesiologists, Soc. of	John T. Stage, Jacksonville	Breckenridge W. Wing, Orlando	" "
Chapter, Am. Coll. Chest Phys.	Alexander Libow, Miami Beach	DeWitt C. Daughtry, Miami	" "
Derm. and Syph., Soc. of	Lewis Capland, Miami Beach	Morris Waisman, Tampa	" "
Health Officers' Society	George W. Edwards, II, Orlando	Lorenzo L. Parks, Jacksonville	" "
Industrial & Railway Surgeons	John H. Mitchell, Jacksonville	William H. Chapman, Jacksonville	" "
Neurology & Psychiatry	W. Tracy Haverfield, Miami	Roger E. Phillips, Orlando	" "
Ob. and Gynec. Society	Dorothy D. Brame, Orlando	J. Champneys Taylor, Jacksonville	" "
Ophthal & Otol., Soc. of	Joseph W. Taylor, Sr., Tampa	Carl S. McLemore, Orlando	" "
Orthopedic Society	Arthur H. Weiland, Coral Gables	Edward W. Cullipher, Miami	" "
Pathologists, Society of	Robert J. Poppiti, Miami Beach	Alfred E. Cronkite, Fort Lauderdale	" "
Pediatric Society	Egbert V. Anderson, Pensacola	J. K. David, Jr., Jacksonville	W. Palm Beach, Nov. 8-9, '52
Proctologic Society	Alex. E. Rosenberg, Miami Bch.	George Williams, Jr., Miami	Hollywood, Apr. 26, '53
Radiological Society	Thomas H. Lipscomb, Jacksonville	A. Judson Graves, Jacksonville	" "
Urological Society	George H. Putnam, Gainesville	David W. Goddard, Daytona Beach	" "
Florida—			
Basic Science Exam. Board	Mr. Paul A. Vestal, Winter Park	M. W. Emmel, D.V.M., Gainesville	Gainesville, Oct. 25, '52
Blood Banks, Association	Donald W. Smith, Miami	Mr. J. M. Potts, Miami	Miami, May, 1953
Blue Cross of Florida, Inc.	Mr. C. Dewitt Miller, Orlando	Mr. H. A. Schroder, Jacksonville	Daytona Beach, Nov. 16, '52
Blue Shield of Florida, Inc.	Leigh F. Robinson, Ft. Lauderdale	Mother Loretto Mary, Tampa	Hollywood, Apr. 26, '53
Cancer Council	Ashbel C. Williams, Jacksonville	Lorenzo L. Parks, Jacksonville	
Dental Society, State	W. G. McLeod, D.D.S., Pensacola	Coleman Brown, D.D.S., Tampa	St. Petersburg, Apr. 20-23, '53
Heart Association	Jere W. Annis, Lakeland	William P. Hixon, Pensacola	
Hospital Association	Mother Loretto Mary, Tampa	Tracy B. Hare, Miami	Daytona Bch., Nov. 17-19, '52
Medical Examining Board	Ralph B. Spires, DeFuniak Springs	Homer L. Pearson, Jr., Miami	Jacksonville, Nov. 23-25, '52
Medical Postgraduate Course	Turner Z. Cason, Jacksonville	Chairman	
Nurses Association, State	Miss Undine Sams, Miami	Bertha King, Tampa	St. Petersburg, Nov. 14-16, '52
Pharmaceutical Association, State	Mr. Clyde Morris, Daytona Beach	Mr. R. Q. Richards, Ft. Myers	St. Petersburg, May, '53
Public Health Association	Mrs. May Pynchon, Jacksonville	Mr. Fred B. Ragland, Jacksonville	Jacksonville, Oct. 9-11, '52
Tuberculosis & Health Assn.	Mr. Walter McJordan, Orlando	Mrs. Elsa B. Conant, Pt. St. Joe	
Woman's Auxiliary	Mrs. Herschel G. Cole, Tampa	Mrs. Herbert B. Lott, Tampa	Hollywood, Apr. 26-28, '53
American Medical Association	Louis H. Bauer, Hempstead, N. Y.	Geo. F. Lull, Chicago	New York, June 1-5, '53
A.M.A. Clinical Session	Louis H. Bauer, Hempstead, N. Y.	Geo. F. Lull, Chicago	Denver, Dec. 2-5, '52
Southern Medical Association	R. J. Wilkinson, Huntington, W. Va	Mr. C. P. Loranz, Birmingham	Miami, Nov. 10-13, '52
Alabama Medical Association	B. W. McNease, Fayette	Douglas L. Cannon, Montgomery	Birmingham, Apr. 16-18, '53
Georgia, Medical Assn. of	C. F. Holton, Savannah	Mr. Sid Wrightsman, Atlanta	Savannah, May 10-13, '53
S. E. Hospital Conference	Mr. Norman Losh, Orlando	Mr. D. O. McClusky, Tuscal'sa, Ala.	New Orleans, April 8-10, '53
Southeastern Allergy Assn.	Clarence Thomas, Nashville, Tenn.	Kath. B. MacInnis, Columbia, S. C.	Havana, Cuba, Mar. 26-29, '53
Southeastern, Am. Urological Assn.	William Miner, Covington, Ky.	Sidney Smith, Raleigh, N. C.	Nashville, May, '53
Southeastern Surgical Congress	H. L. Claud, Washington, D. C.	B. T. Beasley, Atlanta	Louisville, Mar. 9-12, '53
Gulf Coast Clinical Society	Alvin L. Stebbins, Pensacola	Dale E. York, Pensacola	Pensacola, Oct. 16-17, '52



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	SOCIETY	PRESIDENT	SECRETARY	MEETING DATE	MEMBERS Total	MEMBERS Paid	COUNCILOR
A	Bay	Charles H. Daffin, M.D. 536 E. 4th St. Panama City	William F. Humphreys, Jr., M.D. 112 E. 3rd Court Panama City		24	100%	
	Escambia *Santa Rosa*	Chas. J. Heinberg, M.D. 109 N. Baylen St. Pensacola	Raymond B. Squires, M.D. 386 Brent Bldg. Pensacola	2nd Tuesday 8:00 P.M.	76	100%	
	Franklin-Gulf	John W. Hendrix, M.D. Port St. Joe	Photis J. Nichols, M.D. Apalachicola	Last Wednesday	7	100%	A-1-54 Francis [illegible] Watson, M.D. Marianna
	Jackson-Calhoun	Albert E. McQuagge, M.D. 207 N. Green St. Marianna	Francis M. Watson, M.D. 120 Deering St. Marianna	1st Thursday 7:00 P.M. March, June, Sept., Dec.	17	16	
	Walton-Okaloosa	William D. Cawthon, M.D. DeFuniak Springs	Edgar H. Myers, M.D. DeFuniak Springs	3rd Thursday 8:00 P.M.	16	100%	
	Washington-Holmes	L. H. Paul, M.D. Bonifay	Baylye W. Dalton, M.D. Chipley		5	100%	
	Columbia *Baker, Hamilton*	Robert B. Harkness, M.D. 504 E. Duval St. Lake City	Thomas H. Bates, M.D. 27 W. Madison St. Lake City	1st Monday 7:30 P.M.	14	100%	
	Leon-Gadsden-Liberty-Wakulla-Jefferson	Ernest W. Ekermeyer, M.D. 516 N. Adams St. Tallahassee	George H. Massey, M.D. 204 N. Madison St. Quincy	Quarterly 7:30 P.M.	59	55	
	Suwannee	Edward G. Haskell, Jr., M.D. Branford	J. Dillard Workman, M.D. R.F.D. 2, Box 40 Live Oak	1st Saturday 9:30 A.M.	9	100%	A-2-53 Benjamin A. Wilkinson, M.D. Tallahassee
	Madison	A. Franklin Harrison, M.D. Madison	Eugene D. Thorpe, M.D. Madison		3	100%	
	Taylor *Dixie, Lafayette*	Walter J. Baker, M.D. Foley	Ralph J. Greene, M.D. Perry	Last Friday 8:00 P.M.	3	100%	233
B	Alachua *Bradford, Gilchrist, Union*	Henry J. Babers, Jr., M.D. Box 709 Gainesville	Raymond S. Camp, M.D. 907 S. W. 4th Ave. Gainesville	2nd Tuesday 8:00 P.M.	49	48	
	Duval *Clay*	Ferdinand Richards, M.D. 508 Greenleaf Bldg. Jacksonville	Joseph A. J. Farrington, M.D. 415 Greenleaf Bldg. Jacksonville	1st Tuesday 8:15 P.M.	255	243	
	Marion *Levy*	Carl S. Lytle, M.D. 1206 E. Ocklawaha Ave. Ocala	Bertrand F. Drake, M.D. Professional Bldg. Ocala	3rd Tuesday 7:00 P.M.	30	26	B-3-54 William C. Thomas, Jr., M.D. Gainesville
	Nassau	David G. Humphreys, M.D. Fernandina	John W. McClane, M.D. Fernandina	Last Friday 8:00 P.M.	11	100%	
	Putnam	Lawrence G. Hebel, M.D. 119 N. 4th St. Palatka	Bennie J. Massey, M.D. Box 105 Palatka	2nd Tuesday 6:00 P.M.	10	9	
	St. Johns	Charles C. Grace, M.D. 145 King St. St. Augustine	James J. DeVito, M.D. Box 100 St. Augustine	3rd Tuesday 8:30 P.M.	15	100%	
	Brevard	James A. Sewell, M.D. 430 New Haven Ave. Melbourne	Theodore J. Kaminski, M.D. Box 576 Melbourne	2nd Tuesday	23	21	
	Lake *Sumter*	Robert H. Montgomery, M.D. Mount Dora	J. Basil Hall, M.D. Mount Dora	1st Wednesday 7:30 P.M.	26	100%	B-4-53 Eugene L. Jewett, M.D. Orlando
	Orange *Osceola*	Carl S. McLemore, M.D. 1217 Kuhl Ave. Orlando	James B. Glanton, M.D. 1300 Kuhl Ave. Orlando	3rd Wednesday 8:09 P.M.	155	149	
	Seminole	Julian N. Tolar, M.D. First Street Sanford	Frank L. Quillman, M.D. Box 158 Sanford	2nd Tuesday 5:30 P.M.	16	100%	
	Volusia *Flagler*	J. Richard West, M.D. 224 S. Palmetto Ave. Daytona Beach	Robert L. Miller, M.D. 258½ S. Beach St. Daytona Beach	2nd Tuesday 7:30 P.M.	78	77	668
C	Hillsborough	Sherman B. Forbes, M.D. 409 Citizens Bldg. Tampa	James N. Patterson, M.D. 911 Citizens Bldg. Tampa	1st Tuesday 8:00 P.M.	175	169	
	Manatee	John E. Granade, M.D. 114 Walcaid Bldg. Bradenton	Marjorie L. Warner, M.D. 202 Manatee Ave., E. Bradenton	2nd Tuesday 8:30 P.M.	18	16	
	Pasco-Hernando-Citrus	William H. Walters, Jr., M.D. Lacoochee	W. Wardlaw Jones, M.D. Box 247 Dade City	2nd Thursday 7:00 P.M.	16	100%	C-5-53 Hugh G. Reaves, M.D. Sarasota
	Pinellas	John P. Rowell, M.D. 2500 Driftwood Rd., S. E. St. Petersburg	Whitman C. McConnell, M.D. 1517 4th St., N. St. Petersburg	1st Mon. Even Mo. 1st Fri. Odd Mo. 6:00 P.M.	190	186	
	Sarasota	Henry G. Morton, M.D. 555 Golf St. Sarasota	Cecil E. Miller, M.D. 880 S. Orange Ave. Sarasota	2nd Tuesday 8:30 P.M.	44	100%	
	DeSoto-Hardee-Highlands-Glades	Harold S. Agnew, M.D. 705 E. Oak St. Arcadia	Charles H. Kirkpatrick, M.D. Box 389 Arcadia	2nd Tuesday 8:00 P.M.	26	23	
	Lee-Charlotte-Collier-Hendry	John S. Stewart, M.D. Lee Memorial Hosp. Fort Myers	Harvie J. Stipe, M.D. 210 Richards Bldg. Fort Myers	3rd Monday 7:30 P.M.	24	23	C-6-53 Emmett [illegible] Martin, M.D. Haines City
	Polk	Ivan W. Gessler, M.D. 402 Professional Bldg. Winter Haven	Jere W. Annis, M.D. Box 1021 Lakeland	2nd Wednesday 7:00 P.M.	92	89	585
D	Indian River	P. T. McClellan, M.D. Vero Beach	William L. Fitts, 3rd, M.D. Vero Beach Arcade Vero Beach	2nd Tuesday 8:00 P.M.	7	6	
	Palm Beach	Cecil M. Peek, M.D. 535 S. Flagler Drive West Palm Beach	David A. Newman, M.D. 511 Citizens Bldg. West Palm Beach	3rd Monday 8:00 P.M.	116	100%	D-7-53 Erasmus B. Hardee, M.D. Vero Beach
	St. Lucie-Okeechobee-Martin	Richard F. Sinnott, M.D. 209 Koblegard Bldg. Fort Pierce	Adrian M. Sample, M.D. Box 897 Fort Pierce	3rd Thursday 8:00 P.M.	15	13	
	Broward	Lloyd U. Lumpkin, M.D. 918 E. Las Olas Blvd. Ft. Lauderdale	William K. Peck, M.D. 915 N. E. 2nd St. Ft. Lauderdale	4th Tuesday 8:00 P.M.	82	78	
	Dade	Ralph S. Sappenfield, M.D. 630 duPont Bldg. Miami	Nelson Zivitz, M.D. 311 Lincoln Rd. Miami Beach	1st Tuesday 8:30 P.M.	614	579	D-8-53 Donald W. Smith, M.D. Miami
	Monroe	James B. Parramore, M.D. Box 326 Key West	Herman K. Moore, M.D. 600 Elizabeth St. Key West	2nd Thursday 8:00 P.M.	15	14	849

Total 2,335